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ACHALASIA OF THE URINARY TRACT IN CHILDREN.[1]

By J. W. S. LAIDLEY,
Sydney.

ACHALASIA of the various sphincters of the urinary tract is a phenomenon which has received but tardy recognition. The literature on the subject is small, and the ideas of most urologists on the subject are as a rule rather vague. In this article I shall attempt to set down an outline of the problem, in the hope that others may be stimulated by personal observation to contribute further. It is so far by personal observation alone that ideas can be crystallized; but it is certain that in the future valuable experimental work may be done.

Historical.

The history of the recognition of achalasia as a clinical entity is to all intents and purposes a history of the surgical treatment of chronic pyelitis. Chronic pyelitis in female children has always been a common disease, and all children's hospitals constantly have many such patients under treatment. As urology began to be a surgical specialty, attention was focused on these children, whom hospital care and medicines had failed to cure. The first attempt to improve matters was the idea that antiseptics directly introduced into the renal pelvis might turn the scale, so that cures might be effected. To this end the child was submitted to cystoscopic examination and ureteric catheterization, and a small amount of 1% silver nitrate solution was injected into each renal pelvis. It was, of course, also recognized at that time that organic obstruction in any form must be relieved before a cure could be hoped for. For instance, an infected hydronephrosis was not regarded favourably, unless the obstructive element causing the hydronephrosis could be remedied.

By these means many cures were obtained, and the washing out of the renal pelves with silver nitrate solution became an established and fairly successful mode of treatment. What really happened was not that the silver nitrate killed the organisms in the renal pelvis, but that the passage of the cystoscope and the ureteric catheters acted as dilators to those sphincter muscles which were not acting in a coordinated manner. These repeated dilatations relieved the achalasia and the child's condition improved correspondingly.

The next stage came when achalasia of the intestinal tract became better known, and when it was realized that here things could be improved by sympathectomy. Then various attempts were made by such methods as presacral neurectomy and sympathectomy of the renal pedicle to relieve achalasia of the urinary tract. These efforts undoubtedly met with some success, but so far on the whole they have been disappointing. So finally in most cases we are forced back to the original principle of treatment which is often undoubtedly effective—namely, that repeated dilatation of the affected sphincter results in its more coordinated action. Thus it is seen that we appear to be ending where we began. But now we are on sounder ground. We are under no misapprehension any longer about the value of antiseptics introduced as bactericides, and we realize that in the absence of stricture, bladder neck obstruction or urethral deformity, obstruction can still be present. That invisible obstruction is apparently due to failure of coordination of the neuro-muscular mechanism of one sphincter or another, and we term it shortly "achalasia".

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 24, 1942.

Diagnosis.

There is no positive observation by which the diagnosis of achalasia may be made. The sum total of the effects of achalasia is urinary obstruction, and it is only by careful examination that the many more obvious and definite causes of urinary obstruction can be eliminated. When all other causes have been eliminated, then the diagnosis of achalasia may be made. Thus we see that even the approach to the subject of achalasia is difficult, since the diagnosis must be an exclusion diagnosis.

Effects of Achalasia.

The effects of achalasia can be summed up in one word—obstruction.

Of the various urinary sphincters, the bladder neck sphincter is by far the most commonly affected. Mild achalasia of this sphincter will cause frequency of micturition, dysuria or enuresis. Most frequently a child suffering from this form of achalasia will be admitted to hospital with the diagnosis of chronic cystitis, and probably has been undergoing treatment for that condition for some time. On examination the provisional diagnosis can be at once disproved, for no pyuria is present. Then, on cystoscopic examination, the instrument will be found to pass through the urethra without unusual difficulty, and on inspection the mucosa will appear perfectly normal. However, one positive sign of bladder neck obstruction will often be seen; that is trabeculation of the bladder wall. This sign is most important. In the absence of any organic cause for the obstruction, trabeculation must indicate that obstruction is indeed present. So when this sign is seen, one can be confident that the child is suffering from achalasia of the vesical sphincter.

In the next stage of severity, though the bladder wall has hypertrophied still further, it is unable to empty itself completely. At this stage residual urine is present which inevitably sooner or later becomes infected. Now the child is truly suffering from cystitis which resists all forms of medical therapy.

Finally we reach the third stage. In this the bladder is atonic and may contain up to a pint of residual urine, which is usually infected; the ureteric orifices have become dilated and remain permanently open, so that the whole urinary tract is thrown into one cavity, and increase in tension in the bladder means a precisely equal increase in pressure in the renal pelves. Under these conditions bilateral hydronephrosis rapidly occurs and commencing renal failure becomes evident. At this stage, if a cystogram is taken, it will be found that the sodium iodide solution has flowed up the urethra into the renal pelves, and a skiagram will show the whole urinary tract filled with the opaque medium.

Sometimes the sphincter at the junction of the ureter and bladder is affected. When this occurs one of two things may happen. In the ureter and renal pelvis on the affected side, first stasis, and then hydroureter and hydronephrosis will appear, according to the severity of the achalasia. The initial stasis may occasionally be seen very well in excretion urograms. In these the excreted dye outlines the whole ureter, which is not dilated, but which may be visualized in continuity through a series of two or three films. Until infection occurs this condition of hydroureter and hydronephrosis is usually symptomless; but once infection does occur, all the conditions necessary to cause chronic and recurrent pyelitis are present.

Fortunately the milder degrees of achalasia are more common than the severe forms, and consequently the earliest obstructive phenomenon, that of stasis, is most commonly found on investigation.

The rarest form of achalasia is that affecting the muscle bundle at the uretero-pelvic junction. In this form the renal pelvis is the only part affected, the ureter remaining normal. The result before structural changes have taken place is chronic pyelitis; after structural changes have taken place hydronephrosis or pyonephrosis is present. I have often had to remove a hydronephrosis or pyonephrosis, and after the most careful search of the upper end of the ureter for any outside obstructive cause, such as adhesions or a leash of aberrant vessels, I have been unable to find any such obstruction. In this type of case the probable cause of the obstruction is achalasia.

Final Effects of Achalasia.

Mild achalasia, which is fortunately the most common type, tends to disappear spontaneously as age increases, and when no structural changes have occurred the subject is apparently left in a normal condition. The condition becomes increasingly rare as adult life is reached, though I fancy I have seen a small number of young women with urinary disturbances which seem to have persisted since childhood, and which may be due to a long-standing failure of sphincter coordination.

In the less common and more severe types of achalasia structural changes have occurred and tend to be permanent. Now and then a *nullipara* is seen, with a small hypertrophied bladder infiltrated with scar tissue, gaping and dilated ureters and a bilateral hydronephrosis. Usually the urine is heavily infected, and often too a diminution in renal function has occurred. This type of case may be due to severe achalasia if a careful search fails to reveal any obvious cause for the obstruction.

One observation that might well be made here is that poor renal function caused by obstruction at the beginning of life does not seem to respond favourably to drainage in the same way as it does when caused by the obstructive prostate gland at the end of life.

Differential Diagnosis.

To recapitulate: the diagnosis of achalasia is essentially an exclusion diagnosis. It must not be made unless phimosis, pin-point meatus, meatitis, congenital urethral stricture, hypertrophied verumontanum, median bar and valve of the prostatic portion of the urethra have been excluded in the case of the male patient, and unless small meatus, stenosed meatus, vulvitis, chronic urethritis and small urethra have been excluded in the case of the female patient.

Treatment.

In most mild and moderately severe cases the lesion responds well to dilatation of the affected sphincter. This may have to be repeated at intervals; but within a few months nearly all patients can be greatly relieved. This can be demonstrated well in the child who is suffering from chronic and recurring pyelitis; the temperature remains normal for longer and longer periods, the child's general condition improves, and the urine becomes free from pus and organisms.

In severe cases the lesion does not respond well to dilatation, for structural changes are established and seem to be permanent. Drugs such as "Carbachol" and pitressin have their uses, but they should be used when necessary as adjuncts to dilatation. Spinal anæsthesia seems to give relief in some cases, but I have been unable to notice much permanent improvement. Some years ago many children were subjected to presacral ganglionectomy; but the reports have not been very satisfactory or convincing.

Prognosis.

It will be seen from the above statements that in mild cases without structural changes the patients tend to do well, and in a few months a child whose schooling has been repeatedly interrupted by attacks of cystitis and pyelitis may be rehabilitated. I have failed to achieve much in the severe cases. Anatomical changes due to long-standing obstruction seem to be obstinately permanent, even when the obstruction is removed.

Conclusions.

1. Achalasia of the urinary tract in children is not uncommon.
2. The diagnosis can be made only by exclusion.
3. Achalasia is a common cause of chronic and recurrent cystitis and pyelitis, especially in female children.

4. Achalasia affects most commonly the sphincter of the vesical outlet. Less commonly the uretero-vesical and uretero-pelvic sphincters may be affected.

5. In mild cases, in which no structural change has occurred, relief may be given by dilatation of the sphincter involved. In severe cases, in which structural change has resulted, the response to any treatment is not favourable.

6. Achalasia is a disease of childhood and persists rarely into adult life, though structural changes caused by it are permanent and last throughout life.

ACHALASIA OF CHILDHOOD.[1]

By C. H. WESLEY,
Sydney.

There are four conditions to be considered: (i) cardiospasm, (ii) pyloric stenosis, (iii) duodenal ileus (megaduodenum) and (iv) megacolon or Hirschsprung's disease. I have made a survey of admissions to the Royal Alexandra Hospital for Children during a ten-year period, ending June 31, 1941. There was no case which could be considered as one of achalasia in the vicinity of the terminal portion of the ileum, the middle part of the transverse colon, the duodeno-jejunal junction or the pharynx and cystic duct or sphincter of Oddi.

Cardiospasm.

Cardiospasm occurs rarely in later childhood. At or immediately above the diaphragmatic aperture, the circular fibres of the œsophagus are normally contracted, except during deglutition; though they are not thicker than corresponding fibres elsewhere in the tube, they are referred to as the cardiac sphincter. If the sphincter fails to relax, the tube becomes hypertrophied and dilated.

X-ray examination after a barium meal reveals that the œsophagus is greatly dilated and has smooth walls; the distal end has a characteristic "rat tail" shape.

The cardiac sphincter is supplied by the sympathetic and vagus (parasympathetic). Stimulation of sympathetic fibres causes diminution in the peristaltic activity of the œsophageal musculature and contraction of the cardiac sphincter, whereas stimulation of the vagus causes increased peristaltic activity and relaxation of the sphincter. As the left gastric artery carries sympathetic fibres, resection of portion of the artery deprives the sphincter of some of its sympathetic supply (Adson). This treatment has given no results.

Dilatation by the swallowing of bougies loaded with mercury has given the best results. Dilatation through gastrotomy has also given good results.

In looking through the histories of patients suffering from stenosis or atresia of the œsophagus, I found no case which could be placed under the heading of cardiospasm.

Pyloric Stenosis.

In achalasia of the pyloric sphincter, the sympathetic and parasympathetic (vagus) fibres find their way from the cœliac plexus along branches of the cœliac axis. Pronounced thickening and œdema of the pylorus and pyloric antrum is present, and the mucous membrane of the pylorus is thickened and crushed in shape from circular to star shape. This also occurs in the pyloric muscle.

The imbalance is present before birth. The condition is usually brought under view when the mother has left hospital, later than the fourteenth day after confinement. The patient is usually a healthy boy. Constipation may be noticed; the baby vomits occasionally, and then with each feeding the vomiting becomes projectile; the child loses weight, peristaltic waves appear, usually within fourteen to sixteen days, and the baby is very hungry and takes food ravenously. The vomitus contains no bile, except in the rare case in which evidently the pyloric sphincter is not absolutely closed or is relaxed at times.

A small, rounded tumour may be palpated in the right hypochondrium above the level of the umbilicus. Peristaltic waves are sought after a feeding of glucose and water has been given.

A catheter, inserted three or four hours after a meal, will disclose whether the meal has been retained. This is almost diagnostic. A barium meal is retained, and X-ray examination demonstrates pyloric obstruction. A plain X-ray film shows the stomach to be dilated with gas and the intestines beyond the pylorus to be nearly empty.

Differential Diagnosis.

In atresia of the duodenum, the vomitus usually contains bile; the condition appears soon after birth. *Volvulus neonatorum* (non-rotation) associated with colic and rigidity appears soon after birth.

Many children suffering from persistent vomiting are admitted to hospital with a diagnosis of pyloric stenosis, when the actual condition is œsophageal obstruction, œsophagitis, intracranial birth injury, or megaloureter with pyelocystitis.

In pylorospasm muscular hypertonicity is generalized and vomiting is irregular.

Treatment.

"Eumydrin" has been used to treat the condition; but medical treatment should not be persevered with for more than one week if the child is losing weight. Gastric lavage should be carried out, prior to the administration of a meal of milk thickened with cereal. If the child is dehydrated, no time should be lost before the fluid and saline balance is corrected and operation is undertaken.

Rammstedt's operation (1912) is universally carried out through a right paramedian incision, or as by Robertson, of Toronto, through a muscle-splitting operation on the right hypochondrium.

An incision is made through the non-vascular "watershed" on the superior surface of the pylorus and pyloric antrum. The incision terminates at the pyloric vein, which marks the commencement of the duodenum. The incision is carried through the muscle coats which "cut like young celery", as Denis Brown expresses it. The incision must be carried well across the pyloric antrum. The mucous membrane bulges through the incision and must be freed still further with the handle of the knife or with instruments specially devised for the purpose.

The main danger in the operation is that the incision may be carried too far towards the duodenum and the mucous membrane wounded.

The stomach should be deflated before operation; a catheter may be left *in situ*.

Diarrhœa developing after operation is associated with a bad prognosis. This is probably due to infection from other children. These patients do much better if they are isolated. It is held in London that the results obtained by the same surgeons are much better in private hospitals than in general hospitals.

The outlook depends on early diagnosis. The operation on a dehydrated child should be delayed for twenty-four hours for the intravenous administration of saline solution. The prognosis is improved, and better anæsthesia with less risk is assured.

The anæsthetic agent used at the Royal Alexandra Hospital for Children is invariably ether given by the open method, and this is also the anæsthetic agent chosen by Professor Ladd, of Boston. Local anæsthesia is used by some surgeons at the Hospital for Sick Children, Great Ormond Street, London.

After the operation the child is usually fed on sterile water for twelve hours and then on increasing amounts of mother's milk. The mothers are admitted to hospital with the babies. Early operation ensures that the baby will be breast fed.

The operation wound in these cases is apt to open when the stitches are removed. It is a wise precaution to insert several silkworm gut or silk sutures and to consider prescribing vitamin *C*. In long-standing cases vitamin *C* should always be prescribed.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on September 24, 1942.

Points from Clinical Records.

In looking through the histories over a ten-year period to June 31, 1941, I noticed the following points.

The provisional diagnosis was nearly always pyloric stenosis. Other diagnoses were gastroenteritis, rickets, duodenal atresia, starvation and malnutrition. The age of the patients at the time of their admission to hospital varied chiefly from twenty-one to thirty-five days; but in other cases the ages were 42, 56, 77, 80, 112 and in one instance 160 days. Two patients were seventeen days old.

There were 249 cases; 224 of the patients were males and 25 were females.

Projectile vomiting, constipation and oliguria were recorded in nearly every case. Peristaltic waves were present in nearly every case, and a tumour was noted in a large proportion of cases. The child's weight was less than the birth weight.

Frequently the child was the first-born, but in many cases it was the third child.

Blood was found in the urine in three cases.

In the series several members of the same families suffered from pyloric stenosis.

An elevation of temperature up to 103° F., present for two days, was recorded in many cases. Sustained high temperature was associated with a bad prognosis.

Diarrhœa seldom occurred.

The cause of death was noted as cystitis, eventration and peritonitis, pertussis and pneumonia.

It was noted in the histories that a number of wounds had broken down and others were infected. One child was 49 days old and had vomited for 35 days before operation. Many of the patients had been treated as suffering from feeding difficulty.

Early diagnosis was made through the baby health centres and the Tresillian hospitals.

I have examined two recruits who were operated on at the Royal Alexandra Hospital for Children; both said that they had never had indigestion.

Duodenal Ileus (Megaduodenum).

Chronic duodenal ileus may be present without any detectable mechanical obstruction not explained by the relation of the root of the mesentery and superior mesenteric artery to the fourth part of the duodenum.

Epigastric fullness and flatulence after meals may draw attention to the condition. Pain and fullness, nausea, vomiting and headache occur in females of the asthenic type. I found not a single case which could be explained as one of duodenal ileus and achalasia.

Megacolon (Hirschsprung's Disease).

Megacolon is due to achalasia of the anal sphincter, a failure to relax, due to an imbalance of the autonomic nervous system—preponderance of the sympathetic; the sympathetic is over-active or the parasympathetic is under-active.

Many consider that there is a sphincter of the pelvi-rectal junction, the sphincter of O'Beirne. Hurst considered that any obstruction at the pelvi-rectal junction was due to kinking of the dilated and heavy colon at this point, the rectum being more taut and less dilatable.

Since the sphincter is always in a state of spasm, with failure of relaxation, flatus is retained and dilatation occurs up to and including the ileum.

The onward passage of fæces is delayed at the pelvi-rectal junction at the upper valve of Houston. The call to defæcation occurs when fæces find their way into the rectum and distend it. A gastro-intestinal reflex causes peristalsis to pass fæces on from the pelvic portion of the colon into the rectum. The voluntary contraction of the abdominal muscles and diaphragm increases the abdominal pressure and leads to the final reflex in defæcation, when the sphincter relaxes and the passage is opened. When each peristaltic wave reaches the level of the sphincter, the rectum and colon are evacuated. This relaxation fails to occur in Hirschsprung's disease.

The wall of the colon becomes thickened, especially the muscular and submucous layers; there is an increase of muscle fibres and fibrous tissue owing to distension and muscular over-activity.

In megacolon, pyloric stenosis and cardiospasm, many observers have found degeneration of the myenteric plexus (Auerbach's plexus) resulting in an overgrowth of fibrous tissue and abundance of round cells. The colon is increased in length and diameter from the constantly increasing distension of gas, the thick walls of the rectum give way less easily, and gas and fæces fail to enter the rectum. The distended colon reaches the diaphragm and pushes it cranially. The condition is found chiefly in boys, and associated with it are megabladder and megaureter; these are due to failure of the internal sphincter of the bladder to relax owing to sympathetic preponderance.

Distended colon or magacolon has been described as due to an anal ulcer or hæmorrhoids.

The condition occurs chiefly in boys, as opposed to the adult type, which occurs in women and is associated with uterine symptoms. The latter form probably occurs later in life and is not a condition following through from childhood.

Sometimes there are frequent small motions due to scybala, causing retention colitis; but usually increasing constipation is noticed.

The children are listless and very pale. Hurst considered that toxæmia occurred with purgation. In the more advanced cases, pronounced abdominal distension is present. The children occasionally suffer from collapse associated with attempts at defæcation. They suffer from colic and sometimes from right iliac pain and tenderness, probably associated with distension of the caecum and ascending colon.

In "indefinite" appendicitis associated with constipation, it is wise to investigate the condition with a barium enema and X-ray examination; the much rarer megacolon may be found.

On examination, abdominal distension is found, with a tympanitic abdomen. The thickened colon may give the abdomen a "doughy" feeling. *Per rectum*, no abnormality is found. X-ray examination after a barium enema demonstrates the degree of distension of the colon, not the function. X-ray examination after the barium enema has been cleared away will show the capacity of the colon to empty itself and will indicate the severity of the condition. A plain X-ray film reveals a dilated colon.

High spinal anæsthesia will release the sympathetic control and initiate defæcation. The results of the spinal anæsthetic are used to decide the suitability of the case for treatment by sympathectomy. The passage of flatus and a good action of the bowels indicate a favourable prognosis for sympathectomy.

Treatment.

Hurst treated magacolon by dilatation of the anal sphincter and pelvi-rectal junction with specially manufactured bougies and proctoscopes. The size of the dilators and the duration and frequency of the dilatation manœuvres were increased and then decreased. Mothers were trained to carry out the dilatation with bougies. Good results were reported.

Cholines—for example, mecholyl bromide—have been employed. This relieves the constipation. The patients show improvement whilst they are receiving the drug, but the constipation recurs as soon as the drug is suspended. The patients have to be watched by parents and trained to form regular habits of defæcation. Enemata have to be given frequently to empty the colon; paraffin oil emulsions are of value.

It is accepted by most paediatric surgeons that sympathectomy offers the best hope of relief or cure in severe cases; Sir Barrington Ward is enthusiastic about the results of this operation.

The original operation for megacolon was first performed by Sir Robert Wade and Norman Royle in 1926; the operation was first carried out for spastic paralysis, when it was noticed that constipation was relieved.

Four operations are at present in practice. The first is the original operation devised by Sir Robert Wade. The ramisection is carried out extraperitoneally. The approach is made by a large lumbar incision similar to that for nephrectomy. The rami are severed medially to the four lumbar ganglia, and the cord distal to the fourth lumbar ganglion is severed. This procedure is first carried out on the left side, and if the result is not satisfactory, a similar operation is carried out on the right. The rami in this case are posterior to the inferior *vena cava*. In this operation there is also a transperitoneal approach, which is more difficult on account of the distended colon; these patients suffer from shock. With the lumbar approach there is practically no shock, and the patients stand the operation well. The lumbar operation is the operation of choice at the Royal Alexandra Hospital for Children.

The second operation is excision of the presacral nerve. A plexus of nerves consisting of the superior and inferior hypogastric plexuses spreads over the trigone between the common iliac arteries on the sacrum. If this operation is carried out thoroughly, parasympathetic fibres are cut through as they pass cranially from the second and third sacral nerves. The operation is not a justifiable one for boys, as they are rendered sterile though potent. In the female the first stage of labour is said to occur without pain when this operation has been performed; in some cases orgasm is interfered with.

The third operation is the removal of the inferior mesenteric ganglion. To achieve this object it is necessary to clear the sympathetic fibres from around the inferior mesenteric artery at its origin from the aorta. Again, parasympathetic fibres are removed after they have ascended from the second, third and fourth sacral nerves across or behind the left iliac vessels.

The fourth operation is one in which Adson has also divided the splanchnics as they pass through the diaphragm. There is uncertainty about the limits of the distribution of the vagus and sacral autonomics. The upper two lumbar ganglia are excised also in this operation. I will not deal with caecostomy, colostomy and colectomy.

Summary of Cases in Ten-Year Period.

Forty-two patients were treated as suffering from megacolon; sixteen operations were performed. The youngest baby admitted to hospital was three days old, and was suffering from a distended abdomen and diarrhœa. Later, when the child was aged four months, right and left ramisection was performed by Sir Robert Wade. The child was discharged from hospital, relieved. The ages of other patients admitted to hospital were one week, six weeks, two months, three months, five months, seven months, eight months, fifteen months, one year and six months, three and a half years, four years, five years, eight years, nine years, eleven years and twelve years. Many children were in hospital for long periods and were readmitted several times.

The provisional diagnosis was usually megacolon. Other diagnoses were constipation, stenosis of the bowel, pyloric stenosis and rickets, abdominal adhesions with subacute obstruction, gastro-enteritis.

Many of the patients in the lower age groups prior to their admission to hospital were treated as suffering from difficulty in feeding. Many were admitted to hospital *in extremis*—for example, *spina bifida* and pneumonia.

Several of the older children had been subjected to an appendicectomy. The diagnosis was confirmed by X rays in most cases, and in others at autopsy.

The symptoms were invariably distension (sometimes extreme distension) of the abdomen, constipation and some diarrhœa; one patient had a ladder-pattern abdomen, and one had retention of urine for two days.

Deaths were due to pneumonia and whooping cough; one death followed operation.

HEADACHES, TRAUMATIC AND RHEUMATIC: THE CERVICAL SOMATIC LESION.

By MICHAEL KELLY,
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INTRACTABLE headache is to the patient one of the most distressing and to the medical adviser one of the most boring of symptoms. It is boring because it is obscure; and its obscurity is enhanced by the fact that the brain and its membranes, always regarded as the seat of the painful process, are shut up within a bony screen which effectively prohibits observations upon the site of the disorder.

Until recently the subject of headache had met with neglect at the hands of investigators, and the main cause of this neglect was the difficulty in finding an approach to the problem. No subject can be successfully studied without the help of some sort of a key; but in this case such an aid was entirely lacking. Most writers on headache have provided plenty of causes, without discovering any common factor by which the various causes can be related to each other. Thus Forest Dutton (1939) recently wrote a book on the subject, in which he gave a list of more than two hundred causes.

Headache is associated in the popular mind with high blood pressure and tumours of the brain. It is a common symptom of all pyrexial disorders and of many other diseases of a more or less serious nature. It is a symptom of nervous strain, of eye strain, and of sinus disease. One variety—migraine—has achieved a distinctive name. But apart from all these disorders, there will be found many cases in which the chronic headache itself is the disease. Exhaustive investigations provide no clue to the cause; the patient is obviously not dangerously ill, and yet returns time after time to complain of a distressing headache. The harassed medical man finally labels the patient a "chronic" and a nuisance, and consoles himself with the reflection that the pain is of purely nervous origin.

Headaches of Cervical Origin.

In the light of recent investigations, however, it seems certain that an increasingly large proportion of these headaches will be proved to have their origin in the cervical muscles. For many years it has been recognized that a form of fibrositis in the nuchal muscles was likely to cause headache associated with induration in the neck. Osler and McCrae (1923) termed this "indurative headache". Luff (1913), in a Harveian Lecture about thirty years ago, observed that headaches sometimes were caused by fibrositis in the neck. Ray (1934) declared that the pain was caused by fibrositic nodules, which were palpable in the upper borders of the trapezius or complexus muscles. Cyriax (1938) held that rheumatic headache could be diagnosed by the observation that it was worse on hanging the head forwards, when the nuchal muscles were put under strain. Pickering (1939) mentioned that cervical lesions provide one of the commonest causes, and Ussher (1940) claimed that he had relieved severe facial neuralgia by correcting cervical scoliosis and such lesions. Pollock (1941) has stated recently that many so-called neuralgias of the head are instances of rheumatic or indurative headache. Its frequency, he states, varies in different countries in direct proportion to the recognition of its existence. Tender points are found in the neck and at the insertions of the muscles to the occiput; he treats them by long-continued massage. Others who insist on the importance of cervical lesions are Stockman (1940) and Good (1940), who hold that vertigo commonly forms part of the syndrome.

Local Anæsthesia as a Means of Investigation and Cure.

In his earliest reports on the use of local anæsthesia in fibrositis, Kellgren (1938) mentioned a successful case in which the pain of cervical fibrositis had extended up to the occiput. Cyriax also reported favourably, and I was led by these reports to make use of the method in a number

of cases of unexplained headache. Good results were obtained from the start in a proportion of headaches of idiopathic origin, and later I was tempted to try the method, with equally good results, in many cases of post-traumatic headache.

More recently identical results have been obtained with chronic headaches whose cause was recognized—for example, in the headache of high blood pressure or in that which follows cerebro-spinal meningitis. In two earlier papers (1940, 1941) I have reported a few successes; in the present essay I shall summarize my observations on forty successful cases. These represent perhaps a third of the total number investigated.

The subject is a vast one, and many of the observations are necessarily sketchy; but they are brought forward with a firm belief in the importance of the subject, and in the hope that others will be encouraged to make further observations in a field where accuracy of observation is all-important.

Fibrositis in the Neck.

Fibrositis in the neck behaves like fibrositis elsewhere. Thus the pain, which is due to a muscular lesion, is referred to a distance (to the frontal region in many cases). The lesion can be recognized as a localized point of deep tenderness; more widespread secondary tenderness sometimes occurs, which will disappear when the primary lesion is treated with local anæsthesia.

Like fibrositis elsewhere, the cervical lesion may be rheumatic or traumatic in origin, and when the condition is fully established the quality and distribution of the pain may be identical, no matter what the original cause. It may be but one of many lesions in a case of generalized fibrositis, or it may be the only lesion. It may or may not be accompanied by stiffness of the neck. The headache usually is felt in the occipital region of the same side, but very often it is felt in the frontal region as well. Patients often notice that the back of the head is so tender in the mornings that it is uncomfortable to lie on the pillow. In other cases the patient may be quite unaware of the existence of a lesion in the neck, the pain being referred wholly to the front (Cases I and VII). Sometimes the pain is bilateral, and when it is so it usually is due to a single lesion, the pain being referred across the middle line (Cases V and VIII). In these cases the side on which the pain is the more severe is probably the side of the lesion. In every case of chronic headache the neck should be examined for muscular lesions.

I have been unable to confirm Cyriax's observation that lesions may occur in the epicranial tissues. When tenderness of the scalp has been in evidence, I have found it usually to be referred from a lesion situated suboccipitally. As a rule I have found that the more definite the complaint of pain as such, the more likely is a muscular lesion to be found. In addition to pain, giddiness is a common complaint, and it may be almost continuous. Usually, however, the giddiness is felt only when the patient stoops.

As a general rule, I have found that high lesions (above the fourth cervical vertebra) cause headache with or without pain in the neck; while lesions situated lower down are more likely to cause in addition a pain which shoots out to the shoulder. Lesions situated deeply in the suboccipital region often cause no local pain whatever, being referred wholly to the frontal region or the orbit.

Lesions situated more laterally in the neck may be associated with neuralgic pain and tenderness in the temporo-parietal region and the face (Case VI). In almost half of the cases the lesion was situated deeply just lateral to the upper end of the trapezius muscle, in the angle made by it with the posterior border of the sternomastoid (Figures I, II and III).

Reports of Cases.

CASE I.—Trooper R. complained of a pain behind his right eye, of two months' duration. His condition had been investigated by an oculist and a rhinologist, and he had had his antra washed out more than once. There was no history of injury, and the pain was not felt in the back of the head or in the neck. A tender spot was found situated deeply in the right suboccipital muscles (Figures I and II). Infiltration was followed by immediate relief, and four days later he returned to his unit.

CASE II.—Driver C.S.D. had been treated in January, 1942, for fibrositis of the muscles attached to the scapula. At that time he had a slight headache which did not worry him

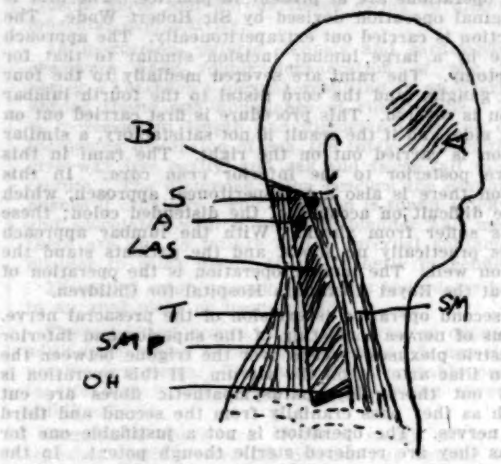

FIGURE I.
S = splenius, LAS = *levator anguli scapulæ*, SMP = *scaleni medius et posticus*, OH = omohyoid, T = trapezius, SM = sternomastoid, A = site of lesion in Case I (common site); oblique shading = site of pain in Case I; B = site of lesion in Case V.

until May, 1942. On June 22 he complained that the headache had been severe for six weeks. It was felt mainly over the right eye, but occasionally spread over the greater part of the head. He was admitted to hospital, but was discharged in a few days, several investigations having given negative results.

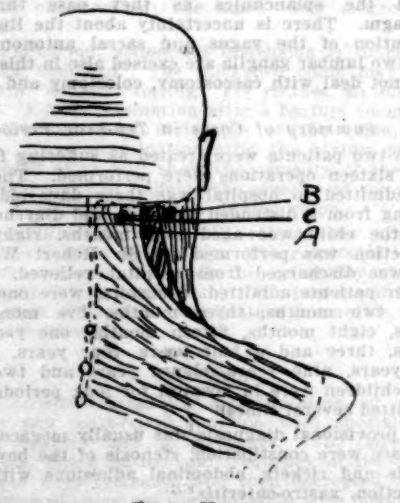

FIGURE II.
A = site of lesion in Case I, B = site in Case V, C = site in Case VIII; horizontal shading = area of referred tenderness in Case VIII.

On June 30 he complained that the pain was more severe. It was noticed that the nuchal muscles on the right side were tender; on pressure over the tender spot the frontal pain was increased. Deep infiltration of this region with five cubic centimetres of "Novocain" solution (0·5%) abolished the pain immediately. On July 3 he complained that the pain had recurred a few hours after the injection. Infiltration of the same spot gave relief once more. On July 7 he had only a mild headache, and the tenderness in the neck was slight. Injection was carried out again, and on July 11 he returned to his unit. Two months later the pain had not returned.

CASE III.—Private McC. complained of a headache of five weeks' duration, felt on both sides of the forehead. On being questioned, he said that the pain was felt in the occipital region too. Neurological and other investigations had revealed no abnormality. On examination, it was found that the nuchal muscles on both sides were tender just below their attachments to the occipital bone. The lesion on the right side was infiltrated; four days later pain was felt only on the left side of the head. The tenderness in the neck was confined to the nuchal muscles on the left side. Infiltration on the left side was followed by a complete cure. A year later he said that the headache had not returned.

CASE IV.—Private R. complained on September 30, 1941, of a severe headache of three weeks' duration, following cerebro-spinal meningitis. The pain was felt above the right eye and at the back of the head. When he stooped the pain became more severe and he felt giddy.

Many investigations had given negative results, and the soldier obviously was suffering severely. On examination, he was found to have a tender area in the right suboccipital region. Deep infiltration gave immediate relief. Four days later he complained only of a slight ache above the eye. There was no pain or tenderness posteriorly, and no dizziness when he stooped. He was transferred to a convalescent depot.

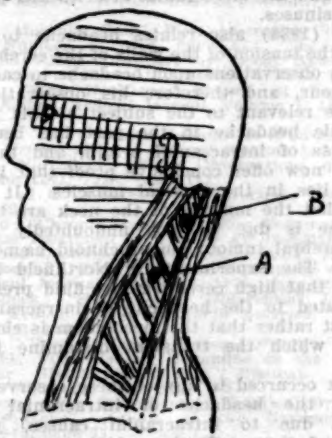

FIGURE III.
A = site of lesion in Case VI; Horizontal shading = area of pain and tenderness in Case VI; B = site in Case VII; vertical shading = site of pain in Case VII.

CASE V.—Private C. complained on October 8, 1941, of a severe headache present ever since the commencement of an attack of cerebro-spinal meningitis three weeks previously. All signs of infection had subsided, and neurological examination gave negative results. The pain was felt behind both eyes and in the neck, being more severe on the right side. The muscles of both sides of the neck were tender, more particularly the right. The tenderest spot was in the posterior fibres of the sternomastoid muscle, one inch below its attachment (Figures I and II). Infiltration of this area abolished all the tenderness on both sides of the neck. Two days later he was sent to a convalescent depot, quite free from headache.

In March, 1942, he complained of pains in the back and elsewhere, on account of which a medical board declared him unfit for the Army. The headache, however, had not returned.

CASE VI.—J.S., aged forty-four years, on March 19, 1941, complained of a severe left-sided headache present since the commencement of an attack of influenza a week earlier. He was suffering much from this neuralgic pain, which would shoot forward across the face. He also complained of a buzzing noise in the left ear and of soreness on the left side of the head. On examination, hyperalgesia of the scalp on the left side and some tenderness of the face were observed. A tender spot was found in the left *levator anguli scapulæ* at the middle of the neck. Infiltration caused the pain and buzzing to disappear immediately, and the relief was permanent (Figure III).

CASE VII.—The patient, an able seaman in the Royal Australian Navy, complained of pain on the left side of the head, mainly in the eye and ear. This had lasted for four weeks, since an accident in which he had been knocked unconscious and had sustained a linear fracture of the skull on the left side. Tenderness was found in the suboccipital muscles on the left side. Infiltration was so effective that he was discharged to the Naval Convalescent Home three days later (Figure III).

CASE VIII.—H.A.N., a seaman in the United States Navy, was examined on July 10, 1942, after he had been under treatment for three weeks. He had sustained an accident, which had rendered him unconscious for a few seconds. Since then he had complained of a constant severe headache, which was growing worse. The pain was felt mainly in the back of the head. His nerves were in a bad condition, and he was made nearly frantic by the ordinary noises of the hospital ward. He could not sit up. All radiological and other investigations gave negative results. The suboccipital muscles were found to be tender on both sides, though perhaps more so on the right.

The tenderest spot was situated only one inch from the middle line, and infiltration was followed by the disappearance of all the tenderness on both sides, which had extended over the back of the head.

On the following day he felt perfectly well, and he was discharged from hospital on July 13 (Figure II).

CASE IX.—Corporal M.W.A. was examined at the same time as H.A.N. His condition was almost exactly similar; he could not sit up, and he was driven frantic by the noise in the hospital. The pain was felt mainly on the left side posteriorly; it had lasted for two weeks, since a fall in which he had knocked his head. A month previous to this he had sustained a head injury and had been in hospital for one week. All investigations gave negative results. On examination, the nuchal muscles on the left side were found to be tender in the angle between the trapezius and the sternomastoid. The injection of three cubic centimetres of a local anæsthetic agent was followed by complete relief, and the patient returned to his unit on July 14, 1942.

CASE X.—Private P. had been sent back from the Middle East because of a severe headache, continuously present for nine months. This was thought to be due to high blood pressure (systolic 160 millimetres of mercury, diastolic 115). The pain was felt in the left occipital region, and on examination the left suboccipital muscles were found to be tender. On January 12, 1942, this area was infiltrated with seven cubic centimetres of "Novocain" solution (0·5%). The headache immediately vanished, and on January 27, at the time of his transfer to the convalescent depot, he had had no more pain. His systolic blood pressure then was 165 millimetres of mercury.

CASE XI.—Mrs. J.C. was examined on September 10, 1941. She had suffered from headaches for several years, following a fall in which she struck the back of her head. The headache was almost continuous, and was accompanied by stiff neck, head noises and giddiness. All possible investigations had been performed, as well as several major operations. The headache was felt on both sides in front and behind, but mainly above the left eye. On examination tenderness was present on both sides of the neck, more especially on the left. Deep infiltration into the suboccipital muscles on the left side was carried out. On the following evening her neck was very stiff. No further headaches occurred until October 15, 1942, when she presented the same clinical picture as at the beginning. The injection was repeated, and she was completely free from headache for six months.

CASE XII.—On January 13, 1942, the patient complained of severe headache and pains in the chest, present for some months. He also complained of "bad nerves" for more than a year, and said that he was losing control of his right leg. The headache was centred mainly in the left eye and the left occipital region. On examination, the eye was red and watery and the upper lid drooped a little. Diplopia occurred when the patient looked to the right. Double Argyll-Robertson pupil was observed. The right leg was spastic, with increased reflexes, clonus and jerky movements. No other neurological signs were observed. The suboccipital muscles on the left side were found to be tender, and the tender spot was infiltrated. Blood was taken for a Wassermann test, which produced a positive reaction. A week later the patient complained only of a slight ache in the right occipital region; there had been no more severe pain on the left side. On January 24 a lumbar puncture was performed, and the left-sided headache returned, though to a mild degree only. A second time it was relieved by treatment of the nuchal muscles, and a month later he had no headache.

The colloidal gold test applied to the cerebro-spinal fluid produced a luetic curve. The pains in the chest were relieved at the first visit by the treatment of muscular lesions in the posterior ends of the intercostal spaces. Like

the headache, they recurred after lumbar puncture and were relieved once more by local anæsthesia.

Analysis of Case Reports.

It is my experience that at least one in every three cases of chronic headache is amenable to immediate cure by local anæsthesia. For the large majority of cures one injection alone has been sufficient. When more than one injection is required, it is regarded as a favourable prognostic sign if appreciable improvement, or an alteration in the character of the pain, has followed the first injection. In the latter case a new lesion will be found in a different situation.

I usually do not persevere if no improvement occurs after three injections at intervals of five to seven days. In many of the cases labelled as failures only one injection was given; but it was deduced from the patients' reactions that perseverance would do no good. In many of the unsuccessful cases no lesion was found in the neck, and therefore no injection was given. In other cases tender areas were found in the nuchal muscles, but they always recurred after injection. In yet other cases the tenderness was abolished without a corresponding improvement in the patient's symptoms.

In the successful cases, so great is the relief given by the injection that the result can be described only as dramatic. This was particularly so in Cases VIII and IX, in which the patients had suffered acutely for several weeks. That each could be cured by a single injection in the space of a few minutes seemed almost incredible.

It is important that this method of cure should be explored in every case of chronic headache; it is equally important that the medical practitioner should realize that failure will occur in more than half of the cases. Though I have not been able to assess with accuracy the factors which render failure more likely, an analysis of the failures shows that certain factors play an important part. Thus extreme chronicity of the lesion is an unfavourable factor, though it does not entirely rule out the possibility of a cure. In most of my successful cases the duration of the headache was less than six weeks. Where the duration is more than six months failure is almost inevitable, though not certainly so (see cases X and XI).

A second unfavourable sign is the presence of localized cervical spondylitis. In the large majority of the cases in which the neck was examined radiologically, no abnormality was found. In a small proportion lipping of some of the vertebræ was seen, and a cure was obtained in none of these. In most of these cases, however, the symptoms were of long duration.

In a large number of cases of post-traumatic syndrome the headache was associated with a painful scar in the supraorbital or frontal region, and in none of these was a cure obtained. It would appear that the prognosis is more favourable when the blow has been struck on the back or the side of the head; the reason probably is that only posterior or lateral violence is likely to cause a lesion of the nuchal muscles.

A final unfavourable sign is nervousness on the part of the patient. When the patient is nearly worn out with long-continued pain, he is pessimistic about all forms of treatment and less inclined to be cooperative. In addition to this, nervous distress seems to play a part in aiding "fixation" of the physical pain, so that it becomes more difficult to eradicate.

Discussion.

No extensive literature has as yet grown up around the subject of this essay. Many of those who have reported success with fibrositis in other parts of the body appear to have passed over the possibility of relieving head pains with local anæsthesia. Cyriax was the first to discuss the subject, and he has been followed by Good and one or two others. Theobald (1941) is one of the latest to report the cure of headaches by local anæsthesia, though he relates his success to anæsthesia of the great occipital nerve. That nerve trunks are not involved is easy to prove, for injections are made at widely separate points in different cases. Furthermore, in no instance have I observed cutaneous anæsthesia to follow the injection, though it is quite common to observe the disappearance of deep tenderness of the scalp. This can be tested by gently pulling on the hairs in the tender area; the suddenness of its return to normal sensitivity is almost unbelievable.

A study of the literature reveals that those who have investigated headache recently have approached the subject from varying angles. Thus Penfield (1927) reports a cure in seven out of nine cases of post-traumatic headache of long standing by insufflation of the meninges with air. His explanation of the headache (and in this he is followed by Pickering) is that the trauma has caused adhesions to develop between the dura and arachnoid, and that these adhesions subsequently become subject to tension.

Pickering has surveyed the mechanism of headaches due to widely varying causes. He used the headache due to histamine and that which follows lumbar puncture as the basis of his investigations, and he relates his findings to the known facts regarding the sensibility of the structures comprising the cranial contents. Pickering is of the opinion that most headaches of intracranial origin are due to tension, and that the regions where tension may produce pain are around the intracranial arteries and possibly also the venous sinuses.

Northfield (1938) also relates headache to "some disturbance in the tension of the walls of the cerebral vessels". He made his observations upon headache in cases of intracranial tumour, and therefore his observations do not appear to be relevant to the subject of this paper. But post-traumatic headache in the past has been regarded universally as of intracranial origin, and the foregoing observations now offer conclusive proof that its origin in some cases lies in the cervical muscles. It is common knowledge that the muscles of the neck are tender when the headache is due to such undoubtedly intracranial causes as cerebral tumour, subarachnoid hæmorrhage and meningitis. The experiments of Northfield have shown conclusively that high cerebro-spinal fluid pressure is not directly related to the headache of intracranial tumour. They suggest rather that the mechanism is closely related to that by which the transient histamine headache is produced.

It has not occurred to any of these observers to reflect that, while the headache of intracranial disease is undoubtedly due to intracranial causes, yet in the mechanism of the production of the pain the cervical muscles may play an important part. In Case XII the headache associated with meningo-vascular syphilis was relieved by the treatment of cervical lesions; when the pain recurred, the same observation was made a second time.

Another who has made important observations on headache is Reid (1940). His work followed that of Pickering, and he reports useful results from the treatment of chronic headache by repeated subcutaneous injections of histamine.

The Nature of Somatic Pain.

It is now a recognized fact, established by many competent observers, that a localized somatic lesion can cause pain which radiates to distant tissues. These tissues, moreover, are tender to deep pressure in many cases, and sometimes the skin becomes hyperæsthetic. This production of tenderness and hyperalgesia Lewis (1937) has compared with a process of secretion. The fact that the reaction can be entirely abolished by anæsthetizing the primary lesion proves that a nervous reflex is concerned in its production.

Not only is local anæsthesia a useful instrument of research, but its use is followed by permanent cure in many cases. This fact has not yet been satisfactorily explained. Perhaps the disintegration of the pain-producing focus interrupts finally a vicious circle, whose function it was by reflex action to produce more and more pain. A great deal of research is required, not only upon the subject of fibrositis in general, but upon the function of somatic structures in the production of headache.

Summary.

1. In headaches due to cervical fibrositis, local anæsthesia has proved a useful method of investigation and cure. In this the pain behaves like the pain of fibrositis elsewhere.

2. Equally good results have been obtained by the application of the method to a number of cases of post-traumatic headache.

3. The lesion in many cases of post-traumatic headache therefore is situated in the cervical muscles rather than in intracranial structures.

4. The suggestion is made that the cervical muscles may play an important part in the mechanism of the production of headaches of truly intracranial origin.

Acknowledgement.

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SHOCK AND THE SYMPATHETIC NERVOUS SYSTEM.

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IN a recent article Sir Hugh Devine (1942) states categorically that "the initiating factors [in shock] are toxic substances shed from the cells of injured tissue", and advocates the theory put forward by Bywaters and Beall (1941) that the anuria and renal changes met with in that form of traumatic shock called by them "crush-injury", are due to some unidentified toxic derivative of crushed or necrotic muscle.

Many weighty considerations militate against the acceptance of such views.

1. It is not possible to establish by cross-circulation or other methods that the blood of an animal suffering from traumatic shock (crushing of the limbs) is toxic (McDowall, 1940).

2. The fall in blood pressure, which follows the release of a tourniquet from an injured limb, is not due to the liberation of toxins from the injured area, but to the sudden enlargement of the vascular bed (McDowall, 1940).

3. The application of a tourniquet to an injured limb, which prevents the return of the venous ("toxic") blood, does not prevent the onset of shock. To prevent shock, the pressure exerted by the tourniquet must at least equal the arterial tension (Blalock, 1930), thus cutting off the blood supply of the limb, and blocking all afferent nerve impulses owing to anoxia (Halliburton and McDowall, 1937).

4. Neither shock nor renal failure is found as a sequel of infarction or absorption of blood clot (tissue necrosis).

5. The renal changes met with in crush-injury are found in cases of injury in which no crushing of muscles has occurred (Mayon-White and Solandt, 1941; Glen, 1941; Young and McMichael, 1941).

6. According to Bywaters and Beall themselves (*loco citato*), the renal damage in cases of crush-injury appears early and is progressive, even though the crushed limb is amputated.

7. A condition due to anoxia, similar to the renal failure of crush-injury, accompanied by similar degenerative changes in the convoluted tubules, is found in the anuria of cholera persisting after successful restoration of the circulation by infusions of saline solution (Tomb, 1942).

Shock has been defined by McDowall (1933) as "the state resulting from a fall in arterial blood pressure which if severe may result in death from oxygen-want (anoxia)".

Sir L. Hill (1941) early suggested that the anuria and renal changes characteristic of so-called crush-injury were due to anoxia.

Anoxia may be caused, apart from circulatory defects and disturbances, by the following factors: (i) by obstruction to the breathing of the normal atmosphere (from strangulation, drowning, pneumonia, asthma *et cetera*); (ii) by diminution of the oxygen content of the inspired air (as at high altitudes or in respiration chambers, and owing to its displacement by other gases, such as carbon dioxide, carbon monoxide, methane, nitrous oxide *et cetera*); (iii) by inhibition of tissue-cell respiration (as in cyanide poisoning); (iv) by diminished oxygen-carrying capacity of the blood: (*a*) from the fixation of hæmoglobin as methæmoglobin, carboxy-hæmoglobin, sulphæmoglobin or nitric-oxide hæmoglobin; (*b*) from diminished blood volume (as in hæmorrhage,[1] collapse in cholera, and traumatic shock);[2] (*c*) from diminished total hæmoglobin content of the blood (as in hypochromic anæmia, hæmolysis, aplastic anæmia and pernicious anæmia).

Bywaters and Beall (1941) report that renal changes similar to those met with in crush-injury are found, together with anuria, in cases of hæmolysis due to incompatible blood transfusion ("transfusion kidney"). Similar degenerative changes of the renal tubular epithelium, with anuria, are also found in blackwater fever (Manson-Bahr, 1935), and in pernicious anæmia (Leftwich, 1920; Stieglitz, 1924); the common factor in these three cases is anoxia due to anæmia consequent on hæmolysis or defective blood formation.

The anuria and renal changes found in crush-injury are therefore manifestations of anoxia due to traumatic shock.

Devine (1942), in enumerating "some of the more important theories of shock" as given by Harkins (1941), fails to mention the theory of over-stimulation of the sympathetic nervous system (Tomb, 1937), though this theory receives the specific commendation of Harkins, and has also received authoritative support from McDowall (1940), who writes of it as follows:

> The possibility that the sympathetic-adrenal system may be overstimulated (in shock) now receives most serious consideration. . . . It is a new idea that [this stimulation] may be harmful or excessive. The possibility is based on the work of Freeman (1933), who showed that if adrenaline is infused into an animal at the rate it is normally secreted the animal passes into a state of shock associated with a concentration of blood and low blood-volume. . . .
>
> Freeman suggests that in the production of shock the sympathetic by its (hyper)-activity causes intense vaso-constriction, a peripheral asphyxia and capillary dilatation, and thus sets up the vicious circle. . . . I am, however, inclined to think that the driving of the blood into the relaxed vessels of the muscles (as suggested by Tomb, 1937) may be just as important and allow loss of blood plasma. . . . It is of special interest to remark that Freeman has found that shock is prevented . . . by removal of the sympathetic or its paralysis by ergotoxine. . . .

[1] Bell (1941) states that Meyler was able to produce uræmia (anuria) readily in guinea-pigs by repeated bleedings.

[2] Anuria and renal insufficiency are sequelæ common to these three conditions (Witts, 1937).

The role of the sympathetic nervous system in shock may be stated as follows. Whenever the body receives any serious injury, an excessive number of afferent stimuli from the injured area reaches the sympathetic nervous system via the brain and spinal cord.[1] The sympathetic nervous system reacts to this over-stimulation by constriction of the cutaneous and splanchnic blood vessels, and by dilatation of the blood vessels of the skeletal muscles; the net result—after, it may be, a preliminary rise—is as a rule a fall in blood pressure. When the stimuli are sufficiently severe to cause the dilator response in the muscle capillaries greatly to outweigh the effect of vasoconstriction in the skin and abdominal viscera, this fall in blood pressure occurs immediately, and symptoms of collapse appear at once (so-called "primary" shock); but when the stimuli are less severe, their effect is cumulative, the fall in blood pressure is progressive, and collapse appears only after a certain interval of time (so-called "secondary" shock).

Owing to capillary dilatation and diminished blood pressure, as well as to complete suspension of muscular activity, capillary stasis ensues. This in time leads to asphyxiation of the capillary endothelium from want of oxygen, with resultant exudation of plasma and loss of blood volume. A vicious circle is thus established, which, if not broken within two or three hours, inevitably ends in death. While this vicious circle in is operation, the introduction of any additional stimuli to the sympathetic nervous system (such as pain, fear, cold, anxiety), as well as the reduction of blood volume by hæmorrhage, sweating or vomiting, will still further increase the state of collapse (Tomb, 1937, 1942).

With regard to the action of adrenaline, Best and Taylor (1940) write as follows:

> In amounts which may be considered (as) physiological . . . adrenaline shows a differential action upon the (blood) vessels. The arterioles and capillaries of the skin, mucous membranes and splanchnic viscera are constricted, except the intestinal vessels which usually dilate, the vessels of the muscles at the same time dilate. With such a dose the constrictor over-shadows the dilator effect, the net result being a rise in blood pressure. The coronaries are dilated. Thus there occurs a redistribution of blood, which is moved from the splanchnic area and skin to the skeletal and cardiac muscles. . . . It is evident from the foregoing that adrenaline does not, as is often assumed, cause general vasocontriction. Certain vessels such as the coronaries, intestinal, and probably also the vessels of the skeletal muscles . . . are dilated even by relatively large doses of adrenaline. The vessels of the skin and of the splanchnic viscera, with the exception of the intestines, are constricted by any effective dose. With very small amounts . . . a fall in blood pressure results. This is attributed to the dilator response in the muscles overbalancing the vasoconstriction in the skin and abdominal organs. . . . On the other hand a depressor (dilator) effect is obtained with large doses if ergotoxine . . . has been given previously. Ergotoxine, it is believed, paralyses the vasoconstrictor mechanism, but leaves the vasodilator intact.

With regard to the therapeutic application of ergotoxine in shock, I have suggested (Tomb, 1937, 1942) the employment of intramuscular injections of ergotoxine, in the doses commonly used in obstetrics—one one-hundred-and-twentieth to one-sixtieth of a grain, repeated if necessary after half an hour—with a view to relaxing the constriction of the blood vessels of the skin and abdominal viscera, and thus assisting in reestablishing the circulation. It may here be pointed out that it is with the object (though unrecognized) of relaxing the constriction of the cutaneous blood vessels that every expedient that will make and keep the patient warm is advocated in the empiric treatment of shock. No expedient, however, for relaxing the spasm of the abdominal blood vessels (Wallace, Fraser, and Drummond, 1917) is suggested in the empiric treatment of shock; this ergotoxine will do (*vide infra*).

It has been found from experience that so-called "vasoconstrictor" drugs, such as adrenaline (which dilates the blood vessels of the skeletal muscles, while constricting those of the skin and abdominal viscera), are not only useless but harmful in shock; the reason is that the effects on the circulation of adrenaline and of shock are identical, owing to the stimulation by the latter of the sympathetic nervous system.

I have had the opportunity of trying the effect of ergotoxine (one-sixtieth of a grain given intramuscularly) in one case only of incipient shock due to crushing of the thumb; the patient was faint, pale and sweating profusely, owing to over-stimulation of the sympathetic nervous system (Tomb, 1942). After ten to fifteen minutes (the physiological time lag when ergotoxine is given intramuscularly) sweating ceased completely and the circulation in the skin, as evidenced by the patient's colour, was restored to normal; although, intentionally, no morphine had been given to him to relieve his pain and to block shock impulses.

I am therefore encouraged to believe that the action of ergotoxine would be still more evident in more serious cases.[2]

Best and Taylor (1940) state that in shock "there is constriction of the arterioles of the splanchnic and cutaneous areas and vasodilatation in the muscles". Apparently, however, they fail to appreciate the true significance of this phenomenon, which is due to sympathetic overstimulation (Tomb, 1937), since they add: "This is probably a compensatory reaction . . . to the unfilled state of the vascular system." On the other hand, they state elsewhere that the unfilled state of the vascular system in hæmorrhage causes a generalized vasoconstriction (an important point which differentiates collapse in hæmorrhage from collapse in shock).

Moreover, as has been pointed out earlier, it is the dilatation of the blood vessels of the skeletal muscles and the consequent stagnation of the blood within them which are the cause of the loss of blood volume as well as of the fall of blood pressure in shock.

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[1] McDowall (1940) states that Beattie and Slome have shown that there is a great increase in the number of impulses passing up the afferent nerves from the injured area.

[2] In profound collapse, when absorption from the tissues is in abeyance, ergotoxine (one one-hundred-and-twentieth of a grain) should, I suggest, be given intravenously.

The Medical Journal of Australia

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CONVERSATION, CRITICISM AND CREDIT.

SILENT people are difficult. The strong silent man, if he exists outside the covers of a novel, may or may not be admired; and silent men are not always "strong". The cynic would say that there was no such being as a silent woman, but he would be in error. The worst of silent people is that you never quite know where you are with them. Does the intent and even superior way in which they look at you bespeak wide knowledge and deep wisdom, either of which would be your undoing if given free play, and are they being merely tolerant of you? Or are silence and austerity merely the camouflage of a vacuous mind? In either case you are in a quandary—you do not wish to be overwhelmed, even potentially, and you do not, if you are really "nice", wish to overwhelm the silent one. In a volume entitled "A Critic in Pall Mall" by Oscar Wilde there appears a review written by him for the *Pall Mall Gazette* in 1887 of a book dealing with the principles of the art of conversation by J. P. Mahaffy. In the volume in question, which is described as a "social essay", Mahaffy stated that every civilized man and woman in society ought to feel it their duty to say something, even when there is hardly anything to be said. But one must beware, for as well as what Oscar Wilde calls the stupidity of silence there is what he so aptly and in alliteration calls the vice of verbosity. In regard to the mental and moral qualifications for conversation Mahaffy held that knowledge was essential, for "an ignorant man is seldom agreeable, except as a butt". On the other hand he thought that strict accuracy should be avoided: even "a consummate liar" was in his opinion a better ingredient in a company than "the scrupulously truthful man, who weighs every statement, questions every fact, and convicts every inaccuracy". In regard to social conversation this is probably correct, for the scrupulously truthful man may, as all recognize, be a frightful bore. Oscar Wilde adds that the liar at any rate recognizes that recreation, not instruction, is the aim of conversation, and is a far more civilized being than the blockhead who loudly expresses his disbelief in a story which is told simply for the amusement of the company.

But this journal is concerned with doctors and medicine, with scientists and science in general. It may be true, as is so often said, that the art of conversation has become lost in general society, and possibly in such circles views like those of Mahaffy might have a useful effect. What can be said of medical and scientific circles? Conversation in general society is usually a matter of making life pleasant by the creation of an amiable and engaging atmosphere. At its best it is both well informed and intelligent. It should be spontaneous and not studied, full of surprise and not dull. Doctors and other scientifically trained persons are as a class said to be able to talk about nothing but their own subject—"shop". If this is so, the terms scientific conversation (or discussion) and conversation of scientists may be looked on as identical. It then becomes useful to inquire in what way scientific conversation differs from that of social life. The chief difference is that scientific conversation is purposive—it is directed to the discovery of new facts, the weighing of evidence, the increase of knowledge. It is largely critical. Of course scientific conversation, like its social opposite, may be on a high level or on a low, it may be good or it may be bad; and its value, its goodness or its badness, depends *inter alia* on the motives of those engaging in it. The proscription of silence does not apply as it may in social conversation, and in fact silence is sometimes desirable and there is no place in it for Mahaffy's "consummate liar". Scientific conversation also cannot have great value among those with illogical minds, those addicted to the production of "red herrings", or those who mount a hobby-horse on every possible occasion. Scientific conversation has at once judicial and creative functions. Its judicial function is apparent, for as already pointed out, it is inherently critical and must sift the grain from the chaff. It is creative, because, like criticism itself, it has to do with interpretation and can inspire those concerned in it to fresh effort.

Scientific conversation is based on observations made by the clinician in the ordinary course of his work, or by a research worker in ward or laboratory; it is thus connected with what is known as credit—some people call it priority. The true scientist is always ready to give credit to those who have made observations in a certain sphere before he has entered it. He will gladly also acknowledge his indebtedness to those who, while working on the same lines as himself, have made observations which he has been able to use. When he himself makes an important observation, perhaps one that completes a research in which many have taken part over a period of years, it is meet and right that he should receive the credit for what he has done; it is reasonable also that he should expect to receive credit. In such circumstances the true scientist is not inflated with pride. He remembers that many before him have brought building material and even laid many stones in the building on which he has been fortunate enough to lay the coping stone. He, moreover, cannot forget that what has been accomplished in science is but a fraction of what remains to be done; and he knows that quite possibly in his scientific explorations he has all but stumbled on some truth which he has passed by because his eyes were not open or his mind alert. Therefore he is humble. Jealousy in a scientist is most distressing. Occasionally it shows its ugly head, for those who harbour it cannot always disguise their failing. They would, if

they could, invariably appear as men of scientific honour and attainment, but they cannot bear the idea that someone may be given certain credit before themselves. It does not really matter if one laboratory is better equipped than another, if one staff chosen by a discerning chief is more enthusiastic and enterprising than another, if certain attractive work falls to the lot of one set of workers rather than to another. It is the discovery of new facts, the laying bare of a hidden secret, that matters. Unfortunately the credit-seekers do not always keep their jealous thoughts to themselves. They may not have the vice of verbosity like Oscar Wilde's social personages, but they do in giving expression to their thoughts bring discredit on science itself. If they cannot readjust their outlook, it were better for them if they became silent. They might then be mistaken for strong silent men of wide knowledge and deep wisdom—if they were lucky.

Current Comment.

DOES ETHER ANÆSTHESIA AGGRAVATE PULMONARY TUBERCULOSIS?

THE belief that ether is an unsuitable anæsthetic drug for patients with pulmonary tuberculosis and that it may greatly aggravate their disease is widely held. H. K. Beecher and R. Adams, of the Massachusetts General Hospital, refer to it as an ill-founded prejudice: they have tried to discover its origin; they have examined the experimental evidence on the use of ether in the presence of pulmonary tuberculosis and they have reported their own experiences with the use of ether for 404 thoracoplastic operations.[1]

It is somewhat surprising to learn that Beecher and Adams were unable to find in the medical literature a single report of the effects of ether anæsthesia in persons suffering from pulmonary tuberculosis based upon a careful study of actual cases. They found, however, much vigorous expression of opinion based upon impressions rather than upon records, and the opinions "varied from the conviction that ether would cure the tuberculosis to the belief that it 'sends them to their graves in less than six months'". The majority of writers who have experimented with animals did not find that ether had any ill effect on pulmonary tuberculous disease; however, nothing daunted, one such author is led from such a negative finding to the suggestion that the rapid spread of the disease which often follows operations on the tuberculous under ether anæsthesia is due to mechanical factors, aspiration from the local lesion aided by laboured respiration and increase of the bronchial secretion diluting the germ-laden sputum. So many writers have shared the view that ether aggravates pulmonary tuberculosis that their statements cannot be dismissed lightly: Beecher and Adams emphasize that most of the articles condemning the use of ether were written from ten to thirty years ago and must have been based on observations of the use of ether by the "open drop" method. They themselves suggest that the coldness of the vapours inhaled when this method is used, the high concentration of ether breathed, which must often be irritating, and the tendency towards asphyxia inherent in the method, all of which disadvantages are overcome today, may have tended to produce ill effects in the patients. They point out that ether has several great advantages over other anæsthetic agents for thoracic surgery, and certainly when administered by them by modern methods it was very well tolerated and their results with it over a period of five years were very good.

There are reasons other than those advanced by Beecher and Adams why serious relapse of pulmonary tuberculosis might possibly follow ether anæsthesia, however administered. It quite often happens that the presence of tuberculosis is unsuspected at the time an operation is performed and subsequent relapse may be attributed to the anæsthetic drug, which is ether more often than not. In such cases symptoms due to the unsuspected tuberculosis could quite easily have been attributed to the disease for which the operation was performed: for instance, septic tonsils may be removed from persons who complained of lassitude, sore throat on swallowing and recurrent "colds"; an operation may be undertaken to correct retroversion of the uterus and other minor abnormalities of the genitalia in young women, perhaps with menstrual irregularities, whose symptoms might not have been obtrusive had the patients themselves not been debilitated and tired and irritable; appendicectomy may be carried out for the relief of chronic appendicitis, a disease the very existence of which is now disputed, in a patient whose pain is referred to the abdomen from the pleura. In all these cases the period of rest in bed after the operation could produce a temporary abatement of the symptoms so that the operation might be deemed to have been successful. Sometimes, as when large numbers of teeth have been extracted, an operation may adversely affect pulmonary tuberculosis by interference with nutrition.

The effects of general surgery and of different kinds of anæsthesia upon pulmonary tuberculosis, latent and manifest, could easily be made the subject of an exact study by anyone attending large numbers of tuberculous patients as in a sanatorium or special hospital. Such a study would be a valuable addition to the work of Beecher and Adams, whose patients were not subjected to a variety of surgical procedures, but underwent particular operations intended to help them in their fight against a known enemy.

DYSMENORRHŒA.

MENSTRUATION is a normal physiological function of woman. A normal function should occasion no pain or even feeling of discomfort. Most women probably regard menstruation as a nuisance whether it produces discomfort or not, but they are not on that account to be regarded as abnormal. As a matter of fact it would probably be correct to assert that a woman would not be normal if she did not look on menstruation as a nuisance. It is also probably true that most women do feel some discomfort and that discomfort may be accompanied by pain of varying severity. If menstruation can be free from suffering of any kind, dysmenorrhœa should be defined as menstruation in which pain or discomfort of any degree is present. To accept any other definition would lead to difficulty. If, for example, dysmenorrhœa was regarded as being present when menstrual pain was so severe that it interfered with the occupation of the sufferer, allowance would have to be made for the varying susceptibility of women to pain and their ability to bear discomfort, as well as for the fact that some women, fewer in number today than they used to be, have no set occupation. That many women contrive to keep their usual activities going in spite of acute discomfort makes the subject of dysmenorrhœa important to the clinician. In the industrial world also the subject is not without its significance, for, at the present time particularly, women are engaged in industry of almost all kinds and the time lost on account of painful menstruation must in the aggregate be considerable. Many years ago R. S. Meaker reported[1] that of 7,876 girls in industrial establishments slightly over 10% sought relief during each month. In one establishment at which 1,750 girls were employed, 96 reported three or more times during eleven months and it was estimated that more than three-fifths of the female members of the staff had occasion during the year to seek advice on account of menstrual troubles. Meaker mentioned an industrial undertaking in which it was the custom to inquire into the

[1] *The Journal of the American Medical Association*, April 4, 1942.

[1] *The Journal of Industrial Hygiene*, June, 1922.

presence or absence of dysmenorrhœa when an employee was engaged and to reject those who admitted regular incapacity. Such establishments must at the present time be few and far between. Helen M. Taylor has recently published a clinical study of menstruation in which she pays particular attention to dysmenorrhœa.[1] Taylor has been concerned with primary dysmenorrhœa only, that is, dysmenorrhœa occurring in the absence of pelvic disease. Her investigation covers 200 patients. Of these 189 were patients on her National Health Insurance list and the rest were private patients. Patients who had been pregnant and those at or about the age of the menopause were excluded. The examination and interview of the women were fairly complete—a general physical overhaul was made and notes were taken of employment, home circumstances, recreation and temperament. The interview which lasted on an average for one and a half hours was supplemented by the fact that Taylor was the regular medical attendant of most of the women—some of them she had attended for as long as eleven years and the average period for the whole series was five and a half years. The youngest of the women was fifteen and a half years of age and the oldest thirty-eight. Twenty-five were under 18 years of age, 125 were between 18 and 25, and 50 were over 25 years of age. Eighty-four were employed in offices, 78 in factories and 19 in shops; 12 were engaged in domestic and six in professional work; one was at school. Twelve were married.

It is important to note that the women were well spread out over the relevant age groups and that the occupations were varied. There is possibly no human function in regard to which superstition has been greater than it has concerning menstruation. Much of the superstition has but recently died, but vestiges still remain. This is important because the mental attitude of the sufferer is such a significant factor in the causation of dysmenorrhœa. At the same time the open air, free and active life of so many Australian young women tends to give them a healthy outlook on all functions of the body. Only 17 of the 200 women in Great Britain ever bathed during menstruation and some of these only did so towards the end of their periods. Many of the women came from homes in which there was no bathroom and a bath once a week was all that was usually taken; this may account for some of the reluctance to take a bath. Taylor gives some further details of lack of cleanliness shown by the women and states that the reasons advanced varied from fear of a chill to fear of "crippling" and mental disease. It is interesting to note in passing that, as shown in the earliest literature, a menstruating woman was regarded as unclean. R. W. Johnstone[2] in a discussion on the subject of menstrual hygiene traced this belief as leading to the custom of segregating the menstruating woman and forbidding her to take part in the ordinary social and religious life of the people. From this view there evolved the belief that menstruation produces temporarily "a condition of peculiar bodily delicacy" which tends to make active exercise undesirable and concentrated mental effort impossible. That superstition does not die easily is shown by the fact that there are in Australia today intelligent women who believe that if a woman while she is menstruating touches a piece of fresh meat, the meat will straightway turn bad. In the face of this kind of thing it is not surprising that dysmenorrhœa presents the clinician with a difficult problem. Though this appears to be a digression, it is not really irrelevant; its introduction at this stage of our discussion is possibly premature.

To return to the facts of Taylor's investigation—it is to be noted that the age of onset of menstruation varied from ten to nineteen years. Few of the subjects had absolutely regular periods. Many healthy girls who were generally regular to within a few days had had periods of temporary irregularity. Taylor divides the sufferers into five grades according to the severity of their dysmenorrhœa. She is careful to emphasize the fact that her series is "loaded with cases of dysmenorrhœa, and gives no information as to the true incidence of menstrual pain and lassitude among women in general". This no doubt robs the observations of much of their didactic value, but for what they are worth the following figures may be quoted. Grade I comprised 30 subjects who had neither local menstrual pain nor discomfort. Grade II comprised 61 subjects who had slight local discomfort sometimes or always. Grade III comprised 48 who usually had a considerable amount of local pain. Grade IV comprised 45 whose pain was "really severe". Grade V comprised 16 whose pain was "extremely severe". Among the more important details noted by Taylor must be mentioned the variability of the pain. It was the rule for pain to vary in intensity from month to month. A number of patients had on more than one occasion manifested both improvement and relapse. Taylor points out that this variation in the severity of dysmenorrhœa from month to month and at different times in the life of the same patient, besides adding to the difficulty of assessing the effect of any treatment, is a characteristic which must be given due weight in any discussion about the ætiology of dysmenorrhœa. A pelvic examination was made in all but four cases. In 127 instances the examination was made *per vaginam* and in 69 instances for varying reasons a rectal examination was made. In not one instance was there any evidence of pelvic inflammation. In 130 patients the uterus appeared to be normal in size and position; in 69 some deviation from normal was found. Details of these 69 cases in which abnormalities were found cannot be given. Since Taylor insists, however, and we think rightly, that surgery ought to have no place in the treatment of primary dysmenorrhœa, it is important to note her observation that it is difficult by clinical examination to decide whether a uterus which shows some deviation from the average size and position may yet be functionally normal. Since under-development and malposition of the uterus are considered by some gynæcologists to be a factor in the production of dysmenorrhœa, Taylor thinks it reasonable to suppose that whatever the nature of the underlying cause, its effect may be modified by local pelvic conditions. This supposition may or may not be justified. If it is, there may be some grounds for the argument that in certain cases of dysmenorrhœa operation to correct malposition should be undertaken. It would be of interest to have records of the experience of Australian gynæcologists in this regard. On the whole it will be safe to look on surgery as having no place in the treatment of primary dysmenorrhœa, in other words to adopt Taylor's dictum.

There are one or two other aspects of Taylor's interesting paper to which reference must be made. The first has to do with hormonal imbalance. She thinks that when methods for the estimation of hormones in the blood and urine are more precise than they are at present, the balance of some or all of them may be found to be disturbed in dysmenorrhœa. Some dysmenorrhœic symptoms suggest an hormonal imbalance, but it does not follow that an endocrine disturbance is the primary cause of the condition or that the correct treatment is the administration of hormones. The variability of the pain and the effects of the different kinds of treatment are not in favour of hormonal imbalance. Taylor also quotes Miller in his contention that when endocrine hypofunction or hyperfunction is mentioned, it is necessary to discover the causes that give rise to disturbance of the endocrine cycle. Much remains to be discovered about the endocrine system and its disorders; future work will probably throw some light on the problem of dysmenorrhœa. The exhibition of endocrine products for menstrual disorders must in the meantime be cautious and properly controlled; in other words only proved knowledge must be put into practice. Finally constitutional and environmental conditions and psychogenic factors must be considered. Of these the latter, already mentioned, are the more important. In the words of B. Venn Dunn[1] the patient must be caught young, before the habit of pain is too well formed. The whole subject of menstruation needs to be freed of taboos and superstition.

[1] *The Journal of Obstetrics and Gynæcology of the British Empire*, August, 1942.
[2] *British Medical Journal*, September 10, 1927.

[1] *British Medical Journal*, June 6, 1931.

Abstracts from Medical Literature.

MEDICINE.

Alkalosis and the Sippy Treatment for Peptic Ulcer.

JOSEPH B. KISSNER AND WALTER LINCOLN PALMER (*Archives of Internal Medicine*, May, 1942) analyse one hundred and thirty-five episodes of alkalosis complicating the Sippy treatment of peptic ulcer. Alkalosis, as indicated by electrolyte changes in the serum, was slight in 71 instances, moderate in 27, and severe in 37 instances; it was found to be not a direct function of the quantity of alkali received, and the electrolyte balance was undisturbed during subsequent alkali therapy in 29 persons who had recovered from a previous episode of alkalosis. Symptoms usually appeared within four to ten days after the onset of treatment, and consisted of a distaste for milk and cream, weakness, dizziness, headache, dryness of the mouth, nausea and vomiting. Many patients with slight alkalosis and several with moderate or severe alkalosis did not complain of any discomfort. There was no constant relation between the severity of symptoms and the degree of chemical alkalosis. The authors state that alkalosis may develop in patients with normal renal function, and that its incidence is apparently greater in the presence of massive hæmorrhage, after the loss of gastric juice either by vomiting or by therapeutic aspiration, and in the presence of preexisting impairment of renal function. They noted that the depression of renal function during alkalosis is usually temporary, although in occasional cases the decrease may persist for several months. Renal function, as indicated by the urea clearance test, is not permanently decreased by the prolonged administration of alkali. For the treatment of alkalosis the authors recommend the discontinuance of alkalis, the administration of either ammonium chloride or, preferably, sodium chloride in amounts varying from five to fifteen grammes daily, and the administration of large amounts of water by mouth or by clysis. Fluids given parenterally, such as physiological or 2% solution of sodium chloride, may be necessary to combat the hypochloræmia in the more severe forms of alkalosis. They prefer physiological solution of sodium chloride to a 5% solution of dextrose in distilled water as supplemental therapy to relieve the dehydration, as it replaces both the lost chloride and water.

Sulphaguanidine.

JOSEPH B. KISSNER, ENID C. RODANICHE AND WALTER LINCOLN PALMER (*The American Journal of Digestive Diseases*, July, 1942) report their experience with the use of sulphaguanidine in non-specific ulcerative colitis and other infections of the bowel. They base their opinions upon the results of treatment of a series of twenty patients, twelve of whom suffered from non-specific ulcerative colitis, two from *lymphogranuloma venereum* of the rectum, two from bacillary dysentery and four from miscellaneous infections of the bowel. The sulphaguanidine was administered in conjunction with the usual therapy employed in each case, and the quanity of the drug used and the duration of treatment varied. In the majority of cases, 10 to 15 grammes of the drug were given daily in divided doses for a period of two to four weeks. The procedure in each case included the examination of the fæces for parasites, anaerobic incubation of the stools for *Bacterium necrophorum*, the use of routine agglutination tests, use of the Frei test, and proctoscopic examination of the rectum and sigmoid. In addition, the following data were obtained every three to four days prior to and during chemotherapy: red and white blood cell counts, hæmoglobin value, differential leucocyte counts; the urine was examined, and the concentration of free and total sulphaguanidine in the blood was determined. Chemical analyses of the crystals obtained from 24-hour collections of urine in several cases were made and in fourteen of the patients studied, bacterial counts of the fæces were made frequently before and during the administration of the drug. The findings of the authors were that sulphaguanidine when given in moderate amounts is absorbed to a much less degree than are other sulphonamides, but that considerable absorption of the drug does occur, after its prolonged use in large quantities. When doses of 10 to 15 grammes are given daily, the blood levels may reach 10 milligrammes *per centum*. Sulphaguanidine is less toxic than other commonly used sulphonamides, which property, the authors state, has been attributed by Richardson to the poor absorption of the drug from the bowel, and by Firor and Poth to its prompt elimination by the kidneys. The authors found that sulphaguanidine, in doses of 10 to 15 grammes daily, usually decreased the bacterial count of the fæces and transforms the flora from one predominantly coliform in type to one composed almost entirely of Gram-positive organisms. The drug was found to be of no value in the treatment of paratyphoid B infection, and to have no advantage over other sulphonamides in the treatment of *lymphogranuloma venereum*. The long-continued use of sulphaguanidine in chronic, non-specific ulcerative colitis has not yielded any striking therapeutic results.

Peptic Ulcer.

J. P. EICHHORN (*The American Journal of the Medical Sciences*, March, 1942) discusses the immediate feeding of patients with bleeding peptic ulcer. The records of 43 patients treated by starvation for one to six days were studied. Of these, eight died, generally about the eighth day. A second series of 37 patients who were treated by Meulengracht's method was reported; of these none died. Since July, 1939, all patients with bleeding ulcers have been treated with frequent nutritious feedings from the time of admission to hospital. Patients who vomited food or blood were refed. Cooked and strained vegetables and fruits were allowed; 100 cubic centimetres of orange juice were given daily; cereals, crackers, wafers, toasted bread, eggs, meat or fish baked, boiled or broiled, cream soups, custards, puddings, jellies, ice cream, and any fluid except coffee were given daily from the beginning. Also 0·7 gramme of ferrous sulphate was given thrice daily, and nine doses of aluminium hydroxide, four cubic centimetres to a dose, were given in 24 hours. Blood transfusions were given (average dose 790 cubic centimetres) when the red cells numbered less than two million per cubic millimetre, when there was evidence of shock or when blood urea nitrogen level was raised. The results were much more satisfactory by this full feeding method. None of the patients who were fed on admission to hospital vomited blood more than twenty-four hours after treatment.

Diabetes Mellitus and Tuberculosis.

TUBERCULOSIS was formerly found to occur in nearly 50% of diabetics. M. F. Mark *et alii* (*The American Journal of the Medical Sciences*, April, 1942) review 349 cases of the combined diseases and conclude that the diabetes usually precedes the tuberculosis. The age of onset of the tuberculosis in diabetics (73·9% after forty years of age in this series) is much later than in non-diabetics. Poorly controlled diabetes predisposes to tuberculosis and causes rapid progression of the disease, whereas effectively treated diabetics are no more subject to tuberculosis than normal persons, and among well-controlled diabetics, the results of treatment compare favourably with those obtained among non-diabetic tuberculous patients. The clinical course of the tuberculosis is very much better when the diabetes is managed with protamine zinc insulin than when regular or crystalline insulin is employed.

Plasma Protein Metabolism.

USING dogs which are healthy and active but present some departure from the strictly normal—anæmia, plasma depletion, bile fistula, Eck fistula—G. H. Whipple (*The American Journal of the Medical Sciences*, April, 1942) has been able to conduct experiments on the production of plasma protein and hæmoglobin particularly in relation to diet. From his results he draws conclusions in plasma protein metabolism. Food proteins yield the amino acids absorbed from the intestinal tract and the amino acids are synthesized in the liver cells (and elsewhere) into plasma proteins. These plasma proteins (and amino acids) supply the protein requirements of the body cells. Normally there is a considerable reserve of plasma protein-forming material (one to five times the circulating mass) and this reserve may be reduced by fasting, low protein diet or plasma depletion. This depletion of protein reserves lowers the body resistance to infection and intoxication. These body protein stores, protein production and protein wear and tear are in a nicely balanced or steady state—a dynamic equilibrium. These proteins can pass readily from plasma into cells and the reverse, without loss of nitrogen. Protein reserves suitable for plasma protein production may be depleted and completely exhausted by fasting, by continued use of protein-free diets or plasma depletion. It may require weeks to deplete the reserves, but once they are depleted, food protein is essential to restore the normal supplies. Serum is the most serviceable form available and three grammes of serum protein by mouth will produce one gramme of new plasma protein in the depleted dog; other foods (liver, beef heart, salmon bread, casein) require four to seven grammes by mouth for

the same result. Cystine appears to be one of the key amino acids in plasma protein building. Infection, liver abnormalities or disease may depress plasma protein production and bring about hypoproteinæmia in spite of adequate food intake. Hæmoglobin in its production may draw on the plasma protein, but hæmoglobin stands apart in the protein economy and does not contribute freely to the protein pool. On the other hand, the body guards jealously the fabrication of hæmoglobin, and given a real need for both plasma protein and hæmoglobin the protein flow favours hæmoglobin, which under these circumstances always is produced in more abundance than the plasma protein.

Meningococcal Encephalitis.

THE involvement of the brain in certain meningococcal infections is emphasized by H. S. Banks and J. E. McCartney (*The Lancet*, February 21, 1942). The step up in virulence and massive infection associated with high epidemic prevalence in 1940-1941 and the rapid clearing of organisms from the blood stream and the meninges produced by the sulphonamide drugs, have contributed to a clearer understanding of the pathogenesis of meningococcal infection. Two types of fulminating encephalitis are described: the adrenal type and the cerebral type, both invariably fatal within twenty-four hours. In the former there is marked hypotension associated with the symptoms of profound toxæmia; in the latter, the symptoms are similar, but coma is always prominent. There is often a combination of these types. The pathological findings, which are described in detail, indicate an overwhelming toxic effect on the brain rather than a bacterial invasion; there is little evidence of actual multiplication of organisms within the brain and this is in striking contrast to the usual form of meningococcal disease. The conclusion is then reached that fulminating encephalitis is a rare form of meningococcal infection produced by the overwhelming toxic effect of massive doses of the most highly virulent organisms in the most susceptible subjects, especially infants. A widespread encephalitis occurs, not only in fulminating but also in rare acute cases; in these there is always purulent meningitis and death occurs in five to seven days. The condition is really an acute encephalomyelitis; adrenal hæmorrhage may be a complication and other focal inflammatory lesions may develop, for example, pericarditis or arthritis. Cases of focal encephalomyelitis are also described, clinically resembling the ordinary focus of meningitis; they are always severe, but lack the deep coma typical of the widespread encephalitis, tending rather to stupor and delirium. The outcome is often fatal, the clinical picture varying widely. The autopsy must include examination of the brain and spinal cord at varying levels to demonstrate the typical lesion which consists of small hæmorrhages in the region of the basal ganglia, mid-brain, medulla and upper cord; capillary thrombosis may also be seen. The meningitis may have cleared completely as a result of sulphonamide therapy and thus the meninges may appear normal. Some marginal encephalitis is usually found in relation to the ordinary forms of meningitis and this is seen as a perivascular polymorphic infiltration around the larger vessels only in the neighbourhood of the meninges; meningococci are more easily demonstrated in these lesions than in the true encephalitis.

Primary Portal Phlebosclerosis.

N. E. REICH (*Archives of Internal Medicine*, January, 1942) urges that the name primary portal phlebosclerosis be applied to the disease in which changes in the venous walls are associated with stenosis of the portal system and portal hypertension, resulting in splenomegaly, and recurrent hæmorrhages into the stomach or lower part of the œsophagus. This disease closely resembles Banti's syndrome, but the onset at an early age, the absence of hepatomegaly, of ascites and of anæmia, and the presence of leucopenia (which may be marked by post-hæmorrhagic leucocytosis) serve to distinguish primary portal phlebosclerosis.

The Iron in Brown Bread.

E. M. WIDDOWSON AND R. A. MCCANCE (*The Lancet*, May 16, 1942), who have studied the iron intake and excretion of persons eating white bread and brown bread respectively, suggest that, despite the large amounts of iron in whole wheat, bread made from it may not be as good a source of iron as is generally supposed.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on September 24, 1942, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. K. S. M. BROWN, the President-Elect, in the chair.

Achalasia of Childhood.

DR. J. W. S. LAIDLEY read a paper entitled "Achalasia of the Urinary Tract in Children" (see page 475).

DR. C. H. WESLEY read a paper entitled "Achalasia of Childhood" (see page 477).

DR. P. L. HIPSLEY thanked the speakers for their interesting and instructive papers, covering a wide field. Dr. Hipsley said that Dr. Wesley had included pyloric stenosis among the achalasias; in Australia it was generally accepted that that condition was due to an achalasia. Dr. Hipsley himself believed that the condition was not due to neuromuscular incoordination at all. The treatment, which was very successful, was simply a linear incision of the pyloric antrum, which in the majority of cases did not include the pyloric sphincter. The fact that that procedure was successful in permanently and completely removing the obstruction seemed to point to some other cause for the condition. Many other theories had been put forward. Achalasia had been put forward; but Dr. Hipsley found it difficult to accept that. If a neuro-muscular incoordination was the cause, he thought it would be logical to attack the condition through the sympathetic nervous system. The pyloric sphincter was in a position where achalasia might be expected. Many babies still died from incessant vomiting, and when examined *post mortem* were found to have no organic defect; that was what would be expected if achalasia was present. Dr. Hipsley thought that such babies, who were considered to have suffered from pylorospasm, might actually have suffered from achalasia. The late Dr. John Flynn, senior, had put forward a theory that in these cases there was a reversion to the type of stomach found in the Edentata. Dr. Hipsley thought that there might be some truth in the theory. With regard to symptoms, Dr. Hipsley said that there was no doubt that if peristaltic waves could be seen, in the vast majority of cases the condition would be pyloric stenosis. There was one other condition that gave rise to characteristic and typical peristaltic waves; that was duodenal ileus, in which there was great dilatation of the stomach and the first part of the duodenum. One other condition, which Dr. Wesley had not mentioned, was obstruction of the œsophagus. At the Royal Alexandra Hospital for Children quite a number of children were seen suffering from repeated vomiting. The obstruction was at the junction of the middle and lower thirds of the œsophagus. At that point a barium meal was held up, and the œsophagus above the obstruction became much dilated. Findlay maintained that the portion of the alimentary tract below the obstruction was really stomach, and that at the junction of the shortened œsophagus and the stomach an achalasia occurred. Dr. Hipsley did not agree with that theory; but many people maintained that such conditions were achalasias. Dr. Hipsley thought that they were due to a congenital narrowing of the œsophagus. Referring to treatment, Dr. Hipsley said that in incising the pylorus for pyloric stenosis he had always used an ordinary ampoule file after cutting through the visceral peritoneum with a knife. An ampoule file was a useful instrument; it was not likely to cut through the mucosa of the stomach. Then it was necessary only to separate the layers with a pair of artery forceps. Dr. Wesley had not mentioned the risk of making an opening into the duodenum. Dr. Hipsley said that sometimes one was too apt to try to extend the incision up into the duodenum; such a procedure was likely to kill the child. Referring to anæsthesia, Dr. Hipsley said that at the Royal Alexandra Hospital for Children they used ether. On reading reports of such cases, he had found that some authorities who had low mortality rates used local anæsthesia or some particular type of anæsthetic agent. At the Royal Alexandra Hospital for Children the mortality rate was about 4%. Dr. Hipsley had never performed the operation using any form of anæsthetic agent but ether given by the "open" method. The anæsthetist was usually a resident medical officer, who was a skilled anæsthetist but not a specialist. There was no doubt that if the children, who were dehydrated, were

given glucose and saline solution before being subjected to operation, the mortality rate should not exceed 3%. Dr. Hipsley remembered one occasion on which the wound broke down. He used to use silkworm gut sutures with an anchor dressing; but he had abandoned that method, and had never had a wound break down since. Dr. Hipsley went on to say that the oldest patient on whom he had operated was aged eleven weeks. The child had been perfectly healthy, and had given no sign of the disorder until the age of about nine weeks; then typical vomiting began and peristaltic waves were visible. At the operation the pylorus was found to be much hypertrophied. Referring to Hirschsprung's disease, Dr. Hipsley said that he did not think that a cure could be expected after any operation on the sympathetic nervous system; the most that could be achieved was improvement in the patient's condition, and that generally did follow sympathectomy. Dr. Hipsley said that there were a number of specimens in the museum of the Royal Alexandra Hospital for Children, obtained from newly born infants whose ureters were enormously dilated, although there was no sign of obstruction at the bladder end. The late Dr. Frank Tidswell always maintained that the condition was one of achalasia occurring before birth. Dr. Hipsley said that he had examined many of these specimens *post mortem*, and no obstruction whatever was to be found.

DR. E. H. M. STEPHEN expressed his appreciation of the successful results achieved by surgeons in the treatment of the achalasias. Several cases he had referred to Dr. Laidley demonstrated this. There had been evidence of stasis with a history of persistent or recurrent pyuria. Careful investigation by Dr. Laidley had led to the discovery of stricture of the urethra by no means marked in degree. Dilatation, which had in some cases needed repeating, had led to a return to complete health. He then referred to the good results obtainable by patient treatment of Hirschsprung's disease. One infant which showed the stunted growth and anæmia associated with this disease had had daily irrigations of the bowel carried out for several years by a mother of less than average intelligence. Her efforts were rewarded as her son became a well-grown, robust boy, well above standard weight and of excellent constitution.

DR. RAYMOND GREEN said that he was in agreement with Dr. Hipsley's opinion that pyloric stenosis was not achalasia. There were quite a number of children who apparently had hypertrophy of the pylorus. Probably some of those children ultimately suffered from typical pyloric stenosis because, owing to bad handling in their early stages, they developed an irritative condition of the stomach and swelling of the mucous membrane and spasm; this ultimately led to a certain amount of block. Often these were children of mothers who had a great deal of milk, and they had been over-fed. Their condition was, of course, put down to pyloric stenosis; but probably if they had been properly handled they would not have developed such a condition. Dr. Green thought that many of the cases of pyloric spasm that were encountered were produced by similar conditions; the sequence might be persistent vomiting, temporary relief obtained by vomiting, but later irritated condition of the mucosa. The stomach was washed out, the diet was graded and other measures were taken; relief was given and the child grew normally. Dr. Green considered that a number of children had a large pylorus; but if they were properly fed and handled they did not develop pyloric stenosis.

DR. D. G. VICKERY said that as Dr. Wesley had described achalasia as a failure to dilate, and as it was realized that failure to dilate was controlled by the sympathetic and parasympathetic nervous systems, he thought that insufficient attention had been paid to the manner in which those nervous systems could be affected by druga. "Eumydrin" was one of the drugs that could deal with achalasia in either the intestinal tract or the urinary tract. Dr. Vickery described the case of a child, aged about seven years, who some years before had swallowed an ordinary picture hook; the hook was situated at the ileo-caecal valve. After six weeks the child was brought to Sydney from the country, as the hook had not been recovered. An X-ray examination showed the hook to be in exactly the same place as before. Dr. Vickery decided to try the effect of belladonna, and after the fourth dose the hook was recovered. Dr. Vickery considered that quite a good demonstration of the point he was making; he had had great faith in belladonna and its derivatives ever since, and had used it in all the milder forms of achalasia. He was sure they were influenced by these drugs. In conclusion, Dr. Vickery thanked the speakers for their papers.

Dr. Laidley, in reply, said that there was only one thing to which he wished to refer. He agreed entirely with Dr. Hipsley that nearly all the more severe forms of urinary achalasia developed *in utero*.

DR. K. S. M. BROWN, from the chair, thanked those who had read the papers and contributed to the discussion.

Correspondence.

INJURIES FROM ANIMALS.

SIR: I am much indebted to the medical men mentioned below for supplementary information to my article on this subject, which appeared in this journal on October 3.

Injury from a Boar.—Dr. A. T. Edwards, Medical Superintendent at Callan Park, Sydney, has kindly supplied the following account of a man who was injured in May, 1926: A certified mental patient, working in the piggery of the mental hospital, Kenmore, New South Wales, was knocked over and attacked by a large boar. The tusks of the latter inflicted a deep wound, some eight inches in length, on the medial aspect of the left thigh, badly lacerating the *adductor longus* and exposing the femoral artery for some inches in the bottom of the wound. To stop the hæmorrhage, the patient plugged the wound with a handful of filth from the pig-yard. Despite this, following a muscle toilet and the application of a pint of hydrogen peroxide before suturing, the wound healed by first intention.

Bites by Rats.—Dr. B. B. Jones, of the Victorian Railways, tells me that the reference to the infant attacked by a rat must almost certainly refer to a patient he attended at Rainsford Street, St. Kilda. He had been called before daylight to see the child. The newspaper report as quoted was substantially correct, though blood was not "streaming from bites", which were superficial and bled little. They fortunately showed no signs of infection and healed with no disfigurement.

Dr. J. C. Harlem, of Pyramid Hill, Victoria, has sent me his notes of the following case which is remarkably like the one just mentioned. He writes:

> In 1937, whilst senior resident medical officer at the Ballarat Base Hospital, Victoria, I was called to the casualty department about 11 p.m. to attend an infant about 8 months old. The mother told me that she had placed the infant in her cot in a room without a light about 8 p.m. Shortly before 11 p.m. she heard the child crying vigorously and went to the room. On switching on the light she was dismayed to see a rat jump out of the cot. The infant had numerous wounds on one cheek; in fact the whole area was covered with small lacerations consistent with what one would expect from the biting of a rat. The greater number of the wounds had penetrated through the dermis. The treatment given was purely local, together with anti-tetanic serum and the child suffered no general ill effects. Unfortunately the mother of the child did not attend the hospital after healing was well on the way and the final result was consequently missed by me.

Captain M. Kelly, of Rottnest, has sent the following three additional examples of injuries received from animals.

Kick from a Horse.—A blacksmith was killed in camp by a kick from a horse on October 17. He was stooping to grasp one of its hind hoofs when it struck him with all its power in the chest with both hoofs. He was thrown a distance of eight feet, became cyanosed and gasped a few times. On Dr. Kelly's arrival within five minutes he was dead. Contusions were visible over the centre of the sternum, but there was no irregularity of the bone to be detected. The coroner did not require an autopsy.

Mussel Poisoning.—The following is Dr. Kelly's own account:

> J.H., a child, aged two, ate some mussels with her parents on the evening of March 8, 1937. During the night she commenced to vomit, and she continued to vomit all the next day and the following evening. On the morning of March 10 she was unconscious, with a temperature of 104°. Vomiting had ceased, and there had been no diarrhœa. She died at midnight. There were no neurological signs, and I regarded the case as one of food poisoning by bacteria. Her father suffered from abdominal pain and nausea on the day following the eating of the mussels.

Painful Stabs from "Cobbler's" (Catfish) Spines and the Value of Local Anæsthesia.—Dr. Kelly's notes are as follows:

A small boy was in great agony after a sting from a "cobbler". The perforation was plainly visible on the sole of his foot. Infiltration of the tissues with 1/1,500 "Percaine" brought immediate relief, and the pain did not return.

In the second case, I myself was inflicted with a painful sting when bathing. As the pain seemed to be getting worse ten minutes later, I infiltrated the tiny perforated wound (on the foot) with "Percaine", 1/1,500. No further pain was felt. An experienced fisherman who saw the wound said that it was the typical result of a cobbler's sting.

I have not known anyone else to use local anæsthesia. Perhaps the method might be of use for spider bites. In the case of the small boy, the pain was felt throughout the limb, and yet it was relieved by a small local infiltration with two cubic centimetres of local anæsthetic.

Injuries Caused by Scorpions.—I regret that, through mislaying the reference, I have omitted to refer to the important paper entitled "Injuries Caused by Australian Scorpions" that Dr. H. Flecker contributed to this journal on June 5, 1937, at page 875. Six cases are described in this paper, so that records of injuries from scorpions in Australia are not, as I stated, remarkably few.

Yours, etc.,

J. B. CLELAND.

University of Adelaide,
Adelaide,
November 3, 1942.

LIGATION OF HÆMORRHOIDS.

SIR: I would like to bring to the notice of your readers a point which I have found useful in the technique of the ligature operation for hæmorrhoids, and which I have not seen mentioned elsewhere.

It consists of the injection of the base of each pile mass (after ligature) with an oil solution of local anæsthetic. I used "A.B.A." (Allen and Hanbury). The result was an entirely painless post-operative period (apart from some flatulent pains), a phenomenon which struck me so that I thought the suggestion worth passing on.

Yours, etc.,

D. N. SHORT.

Adelaide Street,
Blayney,
New South Wales.
November 7, 1942.

"THE CRISIS OF OUR AGE."

SIR: No doubt you will receive more letters than you can possibly print regarding the above article in the issue of the seventh instant. In which case kindly spare us those containing pious platitudes by leading members of the profession.

While the mass of the article (and what a mass!) is not uninteresting, it is the conclusions on which I would like to comment. Your professor's solution of our difficulties is too long to quote in full, but I repeat one sentence: "There must be a concerted preparation for the inevitable mental and moral and socio-cultural revolution of Western society." How this concert of minds is to be achieved he does not say, but I estimate that in order to achieve his object 90% of the world's population would have to be university professors, previously hypnotized so that they would not differ fundamentally among themselves. After this quoted sentence, we are treated to first, second and third steps, each with its "must" and more difficult for poor humanity than all the ten commandments. Finally, with what can only be diabolical irony, he states: "There must *first* be a complete change of mentality and attitude in the light of the doctrine of the Sermon on the Mount." (I underline "first".)

I would like to point out that the Sermon on the Mount has been before the world in spoken and written word for 1,900 years, so that the prospect of achieving its acceptance as a first step is not encouraging. Still, it can be read and understood by most people without doing themselves serious injury from over-study. Not so, however, the professor's part of the programme. I venture to say that not more than 2% of the population of Sydney could read your article, let alone Professor Sorokin's book, without great mental fatigue. As for the medical profession, I put it at 10%.

Now to speak quite seriously. I could tell you why the Sermon on the Mount has not been a practical success—and will do so on request. Perhaps I had better say, briefly, that it is because we are trained from childhood in the art of "getting on in the world" as individuals. Selfishness is at a premium. This is not original sin, it is bad training and bad environment. I am convinced that the time is not far distant when economic changes will occur that will make the Sermon on the Mount acceptable and practical. But I do not think that a mass of philosophical jargon, even in THE MEDICAL JOURNAL OF AUSTRALIA, will help in any way.

I will conclude with an epigram, inspired by your article: The stupidities of an unlearned man are a small matter, but the stupidities of a learned man are colossal.

Yours, etc.,

E. H. STAPLES.

160, Pacific Highway,
North Sydney,
November 9, 1942.

SIR: We are indebted to you for your review of a remarkable and seminal book. Such problems as Sorokin raises are not foreign to us as medical practitioners, for clearly our personal freedom is threatened when men like Dr. Brown can welcome a State medical service with no possibility of private practice outside it, and when our politicians think that they can gull the people of Australia into voting away their constitutional safeguards by accepting an over-riding amendment of the Constitution, such as is embodied in the proposed referendum.

There is an historical antagonism between security and freedom—no nation has achieved both at once, though the endeavour to do so is persistent. The serf is secure, but he buys his freedom whenever it is possible to do so. Then he looks for security and finds himself threatened with a new serfdom. This is the stage we are at now. And correlative with this search for the one or the other or both, goes a change in the mode of government, often finalized by war. We can see for ourselves today that the free nation suffers from unemployment and malnutrition, while the employed nation ("safety first") gets dictatorship and war; so that he who prefers security to freedom ends by getting neither.

Sorokin unfortunately does not consider politics or government as exhibited in his three cultures; nor has he explicitly admitted that war was common to all, and that to do away with war we must have a new synthesis (unless this is what he means by an "integral" culture) of the cultures founded on faith, intuition and the senses, and guided by reason. Sorokin has demonstrated pretty convincingly that we are now in transition from a sensate to an ideational or idealistic culture (he does not know which) and that only by taking thought can we determine our fate. He mentions the inner freedom associated with the ideal cultures and rather seems to despise the outer freedom conferred by the sensate culture. Surely, however, we need both if spiritual (or even material) initiative is to be released, with all its phenomenal possibilities. Certainly our sensate civilization has given us tolerance, freedom from persecution or torture (now returning in its decline) and greater personal freedom, though it has never released our economic bondage (wage-slavery)!

His solution is a civilization based on the Sermon on the Mount—a "familistic" culture; but he puts technique aside. It is hardly fair to ask him to give us the technique, but surely we can expect a statement of the vital principles underlying family life which have to be applied on a national scale if the nation is to live as a great family. Good intentions are not enough—they may, as the proverb says, pave the way to hell. We must choose the correct technique, a technique which must satisfy certain principles.

We can summarize these principles as being first religious (involving the change of heart so often demanded)—obedience to the two Great Commandments, the practice of justice, and kindness, loving one's neighbour as oneself, and bearing one another's burdens. With our attitude right, we then seek the economic principle on which a family lives together—and that is the exchange of work for work, not things for things; while personal responsibility is taken for the sick, the old, the young, and the stranger within (and without) thy gates! This means a new monetary system—herein lies our technique. It exists. May we adopt it!

Yours, etc.,

MARY C. DE GARIS.

Geelong,
November 10, 1942.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1939, of New South Wales, as duly qualified medical practitioners:

McGrath, David Henry, M.B., 1942 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest, Sydney.

McInerney, James Furlong, M.B., B.S., 1942 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

McKell, James, M.B., B.S., 1942 (Univ. Sydney), Royal North Shore Hospital, Saint Leonards.

Maderna, Hector Ernest, M.B., B.S., 1942 (Univ. Sydney), Mater Misericordiæ Hospital, Crow's Nest, Sydney.

Manuel, Pattie Flora, M.B., B.S., 1942 (Univ. Sydney), District Hospital, Balmain.

Maxwell, Clifton Murray, M.B., B.S., 1942 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Michael, Norman Joseph, M.B., B.S., 1942 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Middleton, Lucas William Maxton, M.B., B.S., 1942 (Univ. Sydney), District Hospital, Manly.

Ming, Edna Gork, M.B., B.S., 1942 (Univ. Sydney), Rachel Forster Hospital, Redfern.

Mooney, Austin Jeremiah, M.B., B.S., 1942 (Univ. Sydney), Lewisham Hospital, Lewisham.

Moore, Howard Stewart, M.B., B.S., 1942 (Univ. Sydney), Sydney Hospital, Sydney.

Morgan, David Ross, M.B., B.S., 1942 (Univ. Sydney), Royal North Shore Hospital, Saint Leonards.

Morrison, Ion Craig, M.B., B.S., 1942 (Univ. Sydney), District Hospital, Grafton.

Naval, Military and Air Force.

CASUALTIES.

ACCORDING to the casualty list received on November 18, 1942, Captain J. E. R. Clarke, A.A.M.C., Brisbane, who was previously reported missing, is now reported missing, believed prisoner of war.

According to the casualty list received on November 20, 1942, Major Volney Gordon Bulteau, A.A.M.C., of Gladesville, New South Wales, is reported to have been wounded in action.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Jones, Allan Hackett, M.B., B.S., 1942 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Keston, Frank Keston Cyrus, M.B., B.S., 1941 (Univ. Sydney), Teralba, New South Wales.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association:

Cheek, Nancy Olive, M.B., B.S., 1942 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide, South Australia.

Eyles, Philip Sydney, M.B., B.S., 1942 (Univ. Adelaide), 13, Victoria Avenue, Unley Park, South Australia.

Medical Appointments.

Dr. Horace Joseph Carlyle Edmonds has been appointed, pursuant to the provisions of *The Lunacy Acts* of Victoria, to be Superintendent (acting) of the Mental Hospital, Ballarat, and of the Receiving House, Ballarat, to date from November 3, 1942, during the absence on leave of Dr. James Sydney Alexander Rogers.

The retirement of Dr. James Edward Fancourt McDonald, Medical Superintendent, Toowoomba Mental Hospital, pursuant to the provisions of *The Public Service Acts*, 1922 to 1924, of Queensland, has been cancelled, as from December 31, 1942.

Books Received.

"Prowling through Papua", by Frank Clune; 1942. Sydney: Angus and Robertson, Limited. 8¼" × 5¼", pp. 251, with 16 illustrations. Price: 12s. 6d.

Diary for the Month.

DEC. 1.—New South Wales Branch, B.M.A.: Executive and Finance Committee. Organization and Science Committee.
DEC. 2.—Western Australian Branch, B.M.A.: Council.
DEC. 2.—Victorian Branch, B.M.A.: Annual Meeting.
DEC. 3.—South Australian Branch, B.M.A.: Council.
DEC. 3.—New South Wales Branch, B.M.A.: Special Groups Committee.
DEC. 4.—New South Wales Branch: Annual Meeting of Delegates.
DEC. 8.—New South Wales Branch, B.M.A.: Ethics Committee.
DEC. 8.—Tasmanian Branch, B.M.A.: Branch.
DEC. 9.—Victorian Branch, B.M.A.: Council.
DEC. 10.—New South Wales Branch, B.M.A.: Branch.
DEC. 11.—Queensland Branch, B.M.A.: Annual Meeting.
DEC. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
DEC. 18.—Queensland Branch, B.M.A.: Council.
DEC. 25.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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